U.S. DEPARTMENT OF COMMERCE
National Technical Information Service

PB-254 012

Criteria Documents for Aldrin/Dieldrin

Environmental Protection Agency

June 1, 1976

KEEP UP TO DATE

Between the time you ordered this report—which is only one of the hundreds of thousands in the NTIS information collection available to you—and the time you are reading this message, several *new* reports relevant to your interests probably have entered the collection.

Subscribe to the **Weekly Government Abstracts** series that will bring you summaries of new reports *as soon as they are received by NTIS* from the originators of the research. The WGA's are an NTIS weekly newsletter service covering the most recent research findings in 25 areas of industrial, technological, and sociological interest—invaluable information for executives and professionals who must keep up to date.

The executive and professional information service provided by NTIS in the **Weekly Government Abstracts** newsletters will give you thorough and comprehensive coverage of government-conducted or sponsored research activities. And you'll get this important information within two weeks of the time it's released by originating agencies.

WGA newsletters are computer produced and electronically photocomposed to slash the time gap between the release of a report and its availability. You can learn about technical innovations immediately—and use them in the most meaningful and productive ways possible for your organization. Please request NTIS-PR-205/PCW for more information.

The weekly newsletter series will keep you current. But *learn what you have missed in the past* by ordering a computer **NTISearch** of all the research reports in your area of interest, dating as far back as 1964, if you wish. Please request NTIS-PR-186/PCN for more information.

WRITE: Managing Editor
5285 Port Royal Road
Springfield, VA 22161

Keep Up To Date With SRIM

SRIM (Selected Research in Microfiche) provides you with regular, automatic distribution of the complete texts of NTIS research reports *only* in the subject areas you select. SRIM covers almost all Government research reports by subject area and/or the originating Federal or local government agency. You may subscribe by any category or subcategory of our WGA **(Weekly Government Abstracts)** or **Government Reports Announcements and Index** categories, or to the reports issued by a particular agency such as the Department of Defense, Federal Energy Administration, or Environmental Protection Agency. Other options that will give you greater selectivity are available on request.

The cost of SRIM service is only 45¢ domestic (60¢ foreign) for each complete microfiched report. Your SRIM service begins as soon as your order is received and processed and you will receive biweekly shipments thereafter. If you wish, your service will be backdated to furnish you microfiche of reports issued earlier.

Because of contractual arrangements with several Special Technology Groups, not all NTIS reports are distributed in the SRIM program. You will receive a notice in your microfiche shipments identifying the exceptionally priced reports not available through SRIM.

A deposit account with NTIS is required before this service can be initiated. If you have specific questions concerning this service, please call (703) 451-1558, or write NTIS, attention SRIM Product Manager.

This information product distributed by

NTIS U.S. DEPARTMENT OF COMMERCE
National Technical Information Service
5285 Port Royal Road
Springfield, Virginia 22161

BIBLIOGRAPHIC DATA SHEET

1. Report No. EPA-440/9-76-008

2.

PB 254 012

4. Title and Subtitle
Criteria Documents for Aldrin/Dieldrin

5. Report Date
June 1, 1976

6.

7. Author(s)
Anonymous

8. Performing Organization Rept. No.

9. Performing Organization Name and Address
U. S. Environmental Protection Agency
Office of Water Planning and Standards
401 M Street, S.W.
Washington, D.C. 20460

10. Project/Task/Work Unit No.

11. Contract/Grant No.

12. Sponsoring Organization Name and Address
Office of Water and Standards
U. S. Environmental Protection Agency
401 M Street, S.W.
Washington, D. C. 20460

13. Type of Report & Period Covered
Interim

14.

15. Supplementary Notes

16. Abstracts

This document summarizes the physical/chemical properties, toxicological information and environmental fate and effects of Aldrin/Dieldrin, with emphasis on aquatic behavior. From these data, criteria are developed for the protection of aquatic life and for human exposure.

17. Key Words and Document Analysis. 17a. Descriptors
Criteria
Toxicity
Aquatic animals
Aquatic biology
Human ecology
Safety factor

17b. Identifiers/Open-Ended Terms
Toxic Pollutant Effluent Standards
Federal Water Pollution Control Act

PRICES SUBJECT TO CHANGE

17c. COSATI Field/Group

18. Availability Statement
Release unlimited

REPRODUCED BY
NATIONAL TECHNICAL INFORMATION SERVICE
U. S. DEPARTMENT OF COMMERCE
SPRINGFIELD, VA. 22161

19. Security Class (This Report)
UNCLASSIFIED

20. Security Class (This Page
UNCLASSIFIED

21. No. of Pages
93

22. Price
5.00/2.25

ATTENTION

AS NOTED IN THE NTIS ANNOUNCEMENT, PORTIONS OF THIS REPORT ARE NOT LEGIBLE. HOWEVER, IT IS THE BEST REPRODUCTION AVAILABLE FROM THE COPY SENT TO NTIS.

DIRECT QUESTIONS RESULTING FROM ILLEGIBILITY TO:

Mr. Kent Valentine
EPA (WH 585)
401 M Street, S.W.
Washington, D.C. 20460

Phone: 202-245-3030

ALDRIN/DIELDRIN

TABLE OF CONTENTS

I. Preamble....1

II. Chemical-Physical Properties....5

III. Toxicological Data....8

- a. Microbes....9
- b. Invertebrates....9
- c. Fish....12
- d. Birds....16
- e. Mammals....18
- f. Human Health Hazard....19

IV. Environmental Fate and Effects....21

V. Criteria Formulation....25

VI. References....28

Appendix A

Appendix B

I

INTRODUCTION

CRITERIA DOCUMENTS FOR TOXIC POLLUTANTS

Scientific rationale and criteria developed pursuant to Section 307(a) of the Federal Water Pollution Control Act, P. L. 92-500, 33 U.S.C. §§ 1251 et seq., (1972), for the development and establishment of effluent limitations for toxic substances are set forth in the following chapters.

Section 307(a)(2) states inter alia that a proposed effluent standard "...shall take into account the toxicity of the pollutant, its persistence, degradability, the usual or potential presence of the affected organisms in any water, the importance of the affected organisms and the nature and extent of the effect of the toxic pollutant on such organisms..." Thereafter, having considered these factors, the Administrator is to set an effluent standard for toxic pollutants which provides an ample margin of safety.

In the development of criteria which serve as both the basis and the goal for the establishment of effluent limits, reliance was placed on the toxicity data derived from laboratory studies on a range of organisms including invertebrate, vertebrate, and mammalian test species. These studies provided extensive acute and chronic toxicity data based on feeding experiments for a wide range of aquatic organisms and consumers of aquatic organisms. Environmental studies documenting bioaccumulation in the food web of the toxic material by the food chain organisms and bioconcentration by organisms directly from water provided an important component data base upon which criteria were derived. Appropriate human toxicity data and mammalian carcinogenic studies, where available, were used also in developing criteria.

Aquatic toxicity data generally are obtained by one of two basic methods, the static and flow-through bioassay. The more traditional static bioassay employs a tank in which the test organisms are living and to which a given concentraction of toxicant is added. Any water loss due to evaporation is made up by the addition of fresh water. The flow-through bioassay, which is a more recent development, reflects more nearly the natural conditions. Concentrations of toxic substances are constantly maintained and provide a more accurate test of sensitivity of aquatic species. Water in a flow-through test is replenished constantly through flushing. Comparative results using the static and flow-through bioassays demonstrate that flow-through data yield lower toxicity values for a pollutant than a static bioassay. This fact is demonstrated by comparative studies as discussed in the endrin document. However, most of the data available were developed using static bioassays.

Some toxic pollutants are extremely stable and degrade only slowly or form persistent degradation products. Those pollutants which degrade rapidly pose a less severe long-term hazard unless their entry to the environment is continuous. A parent compound, e.g., aldrin, may rapidly degrade or be altered to a more toxic form, i.e., dieldrin.

Bioconcentration of toxic pollutants is a significant consideration in the development of criterion. The rate and degree of accumulation in an animal and the rate of loss from the animal are factors that help define the potential magnitude of the pollution load problem. As an

1-B

example, a pollutant which bioaccumulates presents a hazard both to aquatic systems and potentially to man or other carnivores associated with the ecosystem. To satisfactorily manage a persistent or non-degradable pollutant requires the maintenance of a ceiling for ambient levels in water which will afford protection to the food chain and the consumers of aquatic life (animals including humans). The body burden of toxic pollutants in fish or food chain organisms may have no outward effect on the species but will affect consumers of that food level. As an example, the brown pelican, when feeding on endrin-contaminate fish may die or suffer species depletion through reproductive impairment.

Data on toxic effects of pollutants are not available for all species that may be exposed to the toxic pollutant in these complex societies. Such data would be necessary to ensure protection of the most sensitive species. It is desirable to know the relative sensitivity of a wide variety of species in order to have a better estimate of the sensitivity of the untested, most sensitive species. Because such data are not available on all species, the range in sensitivity of a small number of tested species is used to provide a measure of the range of sensitivity of all species.

The natural aquatic environment includes many kinds and life stages of plants and animals that are intricately interrelated to form communities. Criteria are developed to protect these interrelationships and incorporate aquatic toxicity data for a phylogenetic cross section of organisms as well as

1·c

species representative of wide geographic distribution. Chronic studies are an important consideration in establishing criteria and require studies of at least one generation, i.e., one reproductive cycle. Use of an application factor for persistent and bioaccumulated toxic pollutants represents consideration of a safety factor. As discussed in the National Academy of Science publication on water quality (p. 185 of the NAS/NAE Water Quality Criteria -- 1972, GPO-5501-00520), the use of an application factor of 0.01 when applied to acute toxic values is thought to provide an ample margin of safety for certain chlorinated hydrocarbon pesticides.

Ecological importance of an organism is dependent on the role the organism plays within the ecosystem and upon its relationship to the food chain within the aquatic community and to consumers of aquatic life, including man. Thus, toxicity data for the top carnivores in a given ecosystem, as well as economically important species such as trout, salmon, menhaden and shrimp are needed for the development of a protective criteria level. Toxicity data for organisms such as the stonefly and Daphnia are of equivalent importance since these organisms are a food base for higher consumers and are representative of invertebrate species found in most waters of the United States.

Invertebrate species, such as the stonefly and the Daphnia, are an indication of the integrity of the aquatic food chain and their presence may be the controlling factor for the abundance of economically and recreationally important predators such as trout, bass or pike. While these fish may not directly consume the *Daphnia* or stonefly or, in fact, even inhabit the same waters, these lower order organisms are representative of the food chain base supporting predators.

Criteria levels, by their nature, are developed to protect aquatic organisms and consumers of aquatic life from direct toxic effect when placed on contact with the toxic pollutants; and, to protect from a more insidious and even greater danger, e.g., chronic effects. Chronic effects take the form of reproductive failures or the poisoning of predators consuming food organisms which have bioaccumulated levels of toxic pollutants as in the case of the brown pelican and consuming endrin loaded fish (see Attachment D, Endrin), and a variety of other physiological effects as discussed in the various documents. Decreases in aquatic organisms or consumers of aquatic life not always are coupled to point source discharges of toxic pollutants at concentrations below acute toxic levels; however, the addition of toxic levels which are not acutely toxic can achieve the destruction or at least disruption of aquatic systems by causing reproduction of failure. Hence, the need for application factors. The relationships between discharges of toxic pollutants and effects on important organisms of economic and environmental importance and consumers of these organisms are well documented in the criteria documents.

An approach to criteria development is to provide ample protection of the test species on the assumption that the response of these species will be characteristic of other associated organisms in the aquatic environment. A number of species have been considered in establishing a criteria

Use of mammalian systems to determine the carcinogenic potential of toxicants found in water follows the same principle as use of aquatic organisms to determine toxicity to fish and other organisms. Carcinogenic substances pose a special hazard to man through environmental exposure. Cancer producing substances may reach man by several distinct pathways.

The following four criteria documents for aldrin/dieldrin, DDT and its metabolites, endrin and toxaphene, represent a survey of the scientific literature documenting the effects of these toxic pollutants to aquatic life and consumers of aquatic life including man. A glossary of terms is provided to define the terms used throughout the documents and will be expanded as necessary when additional documents are added.

1-F

ALDRIN/DIELDRIN

PREAMBLE

Aldrin and dieldrin have been two of the most widely used domestic pesticides. They are related chemical compounds of the chlorinated hydrocarbon family. Although aldrin is used in greater quantity than dieldrin, aldrin quickly transforms into dieldrin in the environment. Hence, there is concern with both compounds. The primary use of the chemicals today is the control of corn pests, although some is used by the citrus industry.

Aldrin use in the U.S. peaked at 19 million pounds in 1966 but dropped to about 10.5 million pounds in 1970. During that same period dieldrin use decreased from 1 million pounds to about 670,000 pounds. The decreases have been attributed primarily to increased insect resistance to the two chemicals and to development and availability of substitute materials (69). Actions to control the use of aldrin/dieldrin were taken by the Environmental Protection Agency as early as 1971. These actions are explained chronologically as follows (69).

In early 1970, based on a concern to limit dispersal of aldrin/dieldrin in the environment, the U.S. Department of Agriculture cancelled all registrations for these pesticides in or on aquatic areas.

On December 3, 1970, the Environmental Defense Fund, Inc. filed a petition with the EPA requesting immediate cancellation and suspension of all Federal registrations of aldrin/dieldrin products under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA), as amended by the Federal Environmental Pesticide Control Act of 1972, 7 U.S.C.S, §135 et seq, on the basis that these substances cause severe environmental damage and are potential carcinogens.

On March 18, 1971, the Administrator of the EPA announced that since the material contained in the petition and in scientific literature raised a substantial question as to the safety of these products, the EPA would issue notices of cancellation of all registrations of aldrin and dieldrin products.

In response to a request by 84 companies whose products would be affected by the cancellation order, a scientific advisory committee reviewed the matter and issued a report in March 1972, recommending that the following uses be disallowed: all applications by aircraft; all foilage methods in which residues are discharged into waterways or setting ponds; all uses in structures occupied by humans or livestock; use on turf except as controlled by trained or licensed pest-control operators, greenskeepers and nurserymen; any use involving application in aquatic environments.

Because the Administrator in 1971 declined to suspend all registrations of aldrin/dieldrin during the pendency of administrative proceedings relating to the cancellation, the Environmental Defense Fund petitioned for review of this decision in the D.C. Court of Appeals.

In May 1972, the Court remanded the matter to EPA for further consideration in light of the advisory committee report, which was issued on May 28, 1972, [Environmental Defense Fund, Inc. v EPA, 465 F. 2d 528 (D.C. Cir. 1972)].

In June 1972, an EPA order lifted cancellation of aldrin/dieldrin for use in deep ground insertions for termite control, nursery dipping of roots and tops of non-food plants, and mothproofing of woolen textiles and carpets where there is no effluent discharge. These are the only registrations being accepted as of the present date. Cancellation of all other major uses of these chemicals was continued, and suspension left open.

During the course of the cancellation proceedings substantial evidence was developed indicating strongly that aldrin/dieldrin pose a severe hazard to human health as well as to the health of other organisms, and that it is a dangerous carcinogen. Accordingly, the Administrator announced on August 2, 1974, his intention to suspend the registrations and prohibit the production for use of all pesticide products containing aldrin or dieldrin which were the subject of the still-pending cancellation proceedings. Following a hearing before EPA's Chief Administrative Law Judge, based upon detailed and extensive findings of fact and conclusions suspension was recommended on September 20, 1974, and was then ordered by the Administrator on October 1, 1974. The Administrator's Notice of Intent to Suspend and Findings as to an Imminent Hazard on August 2, 1974, together with the Recommended

Decision of the Administrative Law Judge of September 20, 1974, and the Administrator's Opinion and Order on the suspension of October 1, 1974, were published in the Federal Register, Vol. 39, No. 203, captioned 39 Fed. Reg. 37246 et seq (October 18, 1974.) Shell Chemical Co. et. al. (EPA FIFRA Docket Nos. 145. etc). A copy of the foregoing i attached hereto as Appendix A.

Thereafter the U.S. Court of Appeals for the D.C. Circuit upheld and affirmed the suspension order, remanding only for the limited purpose of considering whether the ban should be broadened to include existing stocks, Environmental Defense Fund, Inc. v. EPA, 510 F.2d 1292 (D.C. Cir. 1975). A copy of that decision is attached hereto as Appendix B. Among other findings the court upheld the evidentiary basis for the Administrator's conclusions that aldrin/dieldrin are carcinogenic in mice and rats, approved the Agency's extrapolation to humans of data derived from tests on animals, and affirmed the conclusions that aldrin and dieldrin pose a substantial risk of cancer to humans and this constitutes an "imminent hazard" to man.

Regardless of the Administrator's decison as to the cancellation of the various uses of aldrin/dieldrin in the United States, the pesticides will continue to be produced and formulated in this country as long as any uses are permitted and as long as demand for them continues in other parts of the world. Therefore, limits that protect all receiving water uses must be placed on concentrations of aldrin/dieldrin in effluents of plants that produce or formulate these pesticides.

Based upon the data set forth herein, it is concluded that aldrin/dieldrin are harmful to man and aquatic organisms even at very low levels of concentration. It cannot be said that any level of aldrin or dieldrin in the environment is safe, and therefore a prohibition of any discharge is recommended.

I. CHEMICAL-PHYSICAL PROPERTIES

Aldrin (1, 2, 3, 4, 10, 10-hexachloro-1, 4, 4a, 5, 8, 8a-hexahydro-1, 4-endo-5, 8 -exo - dimethanonaphthalene) and dieldrin (1, 2, 3, 4, 10, 10-hexachloro-6, 7 -epoxy-1, 4, 4a, 5, 6, 7, 8, 8a-octahydro-1, 4 -endo -5-8- exo -dimethanonaphthalene) are chemicall related chlorinated pesticides which remain in their toxic form for an indefinite period of time. Physically aldrin and dieldrin are white crystalline substances with aldrin melting at 104°C and dieldrin melting between 176-177°C. Both are soluble in organic solvents with dieldrin the least soluble of the two.

Aldrin is metabolically converted to dieldrin. This epoxidation has been shown to occur in several species including mammals and poultry (1), houseflies (2), locusts (3), soil microorganisms (4), a large number of Lepidoptera species (5), freshwater fish (6), a number of freshwater invertebrates including protozoa, coelenterates, worms, arthropods, molluscs (7), and lobsters (8). The aldrin molecule is biologically altered in the environment to a more stable and at least equally toxic form, dieldrin. Dieldrin is known to be metabolically degraded (9, 89); however, its persistence in the environment is due to its extremely low volatility (i.e., a vapor pressure of 1.78×10^{-7} mm mercury at $20^{o}C$) and low solubility in water (186 ug/l at $25\text{-}29^{o}C$) (10). In addition, dieldrin is extremely apolar, resulting in a high affinity for fat which allows for its retention in animal fats, plant waxes and other non-polar organic matter in the environment. The fat solubility of dieldrin results in the progressive accumulation in the food chain which may result in a concentration in an organism which would exceed the lethal limit for a consumer species.

The affinity of aldrin/dieldrin for animal tissue, a function of low water solubility and the high water partitioning coefficient, shows bioaccumulation is not affected by concentrations of aldrin/dieldrin in water. Many organisms not in direct contact with contaminated water and sediment accumulate aldrin/dieldrin from the food supply. This biological concentration results in tissue concentrations many times those found in the surrounding environment (16). Concentrations increase in the food chain reaching the carnivores at the "top" including man.

Dieldrin is probably the most stable insecticide among the cyclodienes (i.e., isodrin - endrin; heptachlor - heptachlor epoxide). The time required for 95 percent of the dieldrin to disappear from soil has been estimated to vary from 5 to 25 years depending upon the microbial flora of the soil (12). Dieldrin applied at 100 ppm has been shown to persist in soil for more than 6 years (14), while at 25 ppm in a different soil type, a 50 percent loss was found after 7 years (15). When applied to sandy soil at a rate of 100 ppm, residues could be found 15 years later (15). Matsumura and Boush (9) found that of 577 bacterial isolates collected from areas heavily contaminated with dieldrin, 10 isolates would alter dieldrin to 2 to 9 unknown metabolites. The microbes were members of Pseudomonas, Bacillus and Trichoderma genera. Subsequent microbiological studies have revealed that Aerobacter aerogenes also will alter dieldrin similar to 6, 7- trans-dihydroxydihdroaldrin (13). Chacko, et al. (11) tested the ability of 17 species of fungi and actinomycetes. Though most degraded PCNB or DDT or both, none degraded dieldrin.

Patil and co-worker in 1972 studied the metabolic transformations of aldrin/dieldrin by marine algae, surface film, sediments, and water. They found the insecticide was not degraded or metabolized in sea water or polluted waters. Some marine algal populations have been shown to degrade aldrin to dieldrin (89).

Alteration of dieldrin by bacterial systems results in the formation of at least one acidic product (9). Once in the fatty tissue of organisms, dieldrin remains stable (16). However, dieldrin can be mobilized from fatty tissue; for example, when fish are placed in an environment without dieldrin, there is an elimination from the tissue (17). The elimination rate depends upon the diet with fasted fish eliminating dieldrin more rapidly than from fed fish because of the utilization of fat stores (18). The dieldrin eliminated from the tissues reenters the water and thus become available for bioconcentration by other organisms. The movement of dieldrin among organisms, water, and sediment is dynamic, with equilibrium attained when the chemical concentration is constant.

II. TOXICOLOGICAL DATA

Toxic effects resulting from the presence of aldrin/dieldrin in water have been documented for aquatic organisms representing a wide phylogenetic cross section and geographic distribution. While all test organisms used may not be universally distributed in the waters of the United States, they represent types of organisms present in fresh, marine and estuarine systems throughout the country. Extrapolation from the effects found in laboratory and field tests is a reliable means for predicting effects of aldrin/dieldrin on individual organisms and their food chains and is recognized as such by the scientific community.

It should be noted that LC50 values reported for static tests are likely to be substantially higher than LC50 values found using flow-through bioassays. For instance, Earnest *et al.* (93) reported both static and flow-through 96-hr aldrin TL50 (LC50) values for two species of surf perch, *Cymatogaster aggregata* and *Micrometrus minimus*. The former yielded a static value of 7.4 ug/l and a flow-through value of 2.26 ug/l, while *Micrometrus minimus* yielded a static TL50 (LC50) of 18.0 ug/l and a flow-through TL50 (LC50) of 2.03 ug/l. These data suggest loss of toxicant in static bioassays. Static tests in which dissolved oxygen and toxicant concentrations are measured periodically are more reliable than those in which these parameters are not monitored. The flow-through bioassays more accurately reflect nature, where "container wall" effects are likely to be negligible and where the volume of water per fish is much greater.

Toxicological data show aldrin/dieldrin to be acutely toxic to aquatic invertebrates(27), to fish (91, 93), to birds (46), and to mammals (61), although mammalian acute toxicity is relatively low.

a) Microbes

Information regarding the effects of aldrin/dieldrin on bacterial and algal populations is limited. Bacterial species carrying out the conversion of ammonia to nitrate were inhibited when aldrin concentrations were between 100,000 to 10,000,000 ug/kg (21). Studies of the effect of dieldrin on soil bacteria demonstrated a reduction in the numbers of bacteria at soil pesticide concentrations of between 100 to 1000 ug/l. Recovery of bacterial populations varied depending upon species, and required from 7 to 28 days to reach pretreatment numbers and diversity (23). Similarly, diatom species are able to survive concentrations of dieldrin greater than invertebrates and vertebrates. A 50 percent reduction in the diatom population resulted from an application of 12,800 ug/l dieldrin (22). However the growth rate of four types of marine phytoplankton has been reduced 50 percent by dieldrin concentrations ranging from about 100 ug/l to about 500 ug/l (32).

b) Invertebrates

Sensitivity of invertebrate organisms to aldrin-dieldrin is several orders of magnitude greater than that of microbial organisms. Dieldrin distributed at 1 lb/acre in a Florida marsh resulted in a complete annihilation of the crab population and virtual elimination of other crustaceans (24).

Aldrin and dieldrin toxicity to invertebrates is seen in Table 1 and Table 2, and indicate that both compounds are extremely toxic to arthropods. Sensitivity of the stonefly, *Agroneuria pacifica*, to dieldrin in a 20-day continuous flow bioassay system was 0.2 ug/l (37). A sensitive marine crab, *Leptodius floridanus*, exhibited delay in development at concentrations of 1 and 0.5 ug/l dieldrin (38). At 0.9 ug/l dieldrin there was a 55 percent mortality in commercially valuable pink shrimp, *Penaeus duorarum*, within 96 hours (34). Dieldrin is more toxic than aldrin to the insect, *Pteronarcys californica*, and the crustacean, *Gammarus lacustris* (30). Aldrin has been shown to be more toxic than dieldrin to the crustacea, *Simocephalus serrulatus*, and *Daphnia pulex* (30, 31).

Table 1

Acute Toxicities for Various Invertebrates Exposed to Dieldrin

Species	Exposure Time (hr)	Method	LC50 (ug/l)	Ref.
Stonefly (Pteronarcella badia)	24	S	3	27
Stonefly (*Claassenia sabulosa*)	24	S	4.5	27
Stonefly (*Pteronarcys californica*)	24	S	6	27
Stonefly (*Pteronarcys californica*)	48	S	1.3	27
Stonefly (*Pteronarcys californica*)	96	S	.5	27
Amphipod (*Gammarus lacustris*)	24	S	1400	29
Waterflea (*Simocephalus serrulatus*)	43	S	240(EC50)	31
Waterflea (*Daphnia pulex*)	48	S	250(EC50)	31
Grass Shrimp (*Palaemonetes pugio*)	96	F	8.64	34
Pink Shrimp (*Penaeus duorarum*)	96	F	0.7	34
Brown Shrimp (*Penaeus aztecus*)	24	F	25(EC50)	90
Brown Shrimp (*Penaeus aztecus*)	48	F	5.5(EC50)	90
Sand Shrimp (*Crangon septemspinosa*)	24	S	58	28
Sand Shrimp (*Crangon septemspinosa*)	48	S	10	28
Sand Shrimp (*Crangon septemspinosa*)	96	S	7	28

S Static Bioassay
F Flow-through Bioassay

Table 2

Acute Toxicity for Various Invertebrates Exposed to Aldrin

Species	Exposure Time (hr)	Method	LC50 ug/l	Ref.
Amphipod (*Gammarus lacustris*)	24	S	45	29
Sand Shrimp (*Crangon septemspinosa*)	24	S	30	28
Sand Shrimp (*Crangon septemspinosa*)	48	S	14	28
Hermit Crab (*Pagurus longicarpus*)	24	S	300	28
Hermit Crab (*Pagurus longicarpus*)	96	S	33	28
Stonefly (*Simocephalus serrulatus*)	48	S	23(EC50)	31
Waterflea (*Daphnia pulex*)	48	S	28(EC50)	31

c) Fish

When exposed to dieldrin at 1.35 ug/l for four days, estuarine fish, *Leiostomus xanthurus*, were found to have degenerative changes in gills and visceral tissue (34). Growth rates and reproductive performance of the sailfin molly, *Poecilia latipinna*, were adversely affected during a 34-week exposure to 0.75 ug/l dieldrin (35).

Cairns *et al.* (36) found that in the first two or three months of exposure to concentrations of dieldrin ranging from 1.3 to 10 ug/l guppy populations increased more among exposed groups than among controls. The authors attributed this to decreased predation by adult fish on the young. After about six months this population difference disappeared, apparently because the exposed groups were less successful reproductively. Growth rates of rainbow trout, *S. gairdneri*, were reduced by dieldrin concentrations in water of 0.12 ug/l and above, but eggs survived at concentrations of 52 ug/l (42). The LC50 for various fish species is seen in Table 3 for aldrin and in Table 4 for dieldrin.

Table 3

Toxicity of Aldrin to Various Fishes

Species	Exposure Time (hr)	Method	LC50 ug/l aldrin	Ref.
Rainbow trout (*Salmo gairdneri*)	48	U	31	44
Bluegill (*Lepomis macrochirus*)	96	U	5.2	44
Goldfish (*Carassius auratus*)	96	U	28	40
Atlantic silverside (*Menidia menidia*)	24	S	45	91
Atlantic silverside (*Menidia menidia*)	48	S	20	91
Atlantic silverside (*Menidia menidia*)	96	S	13	91
Juvenile White Mullett (*Mugil curema*)	48	F	2.8(Tlm)	90
Bluehead (*Thalassoma*)	24	S	15	91
Bluehead (*Thalassoma*)	96	S	12	91
Striped Killifish (*Fundulus majalis*)	24	S	58	91
Striped Killifish (*Fundulus majalis*)	48	S	26	91
Striped Killifish (*Fundulus majalis*)	96	S	17	91

Cont. of Table 3

Toxicity of Aldrin to Various Fishes

Species	Exposure Time (hr)	Method	LC50 ug/l Aldrin	Ref.
Striped mullet (*Mugil cephalus*)	24	S	126	91
Striped mullet (*Mugil cephalus*)	48	S	100	91
American eel (*Anguilla rostrata*)	24	S	18	91
American eel (*Anguilla rostrata*)	96	S	5	91
Mummichog (*Fundulus heteroclitus*)	24	S	22	91
Mummichog (*Fundulus heteroclitus*)	96	S	8	91
Northern Puffer(*Sphaeroides maculatus*)	96	S	36	91
Bluehead (*Thalassoma bifasciatmm*	96	S	12	91
Striped Bass (*Morone saxatilis*)	96	F	7.2(TL50)	92
Shiner Perch (*Cymatogaster aggregata*)	96	S	7.4	93
Shiner Perch (*Cymatogaster aggregata*)	96	F	2.26	93
Dwarf Perch (*Micrometrus minimus*)	96	S	18.0	93
Dwarf Perch (*Micrometrus minimus*)	96	F	2.03	93

S Static Bioassay
F= Flow-through Bioassay
U= Unknown

Table 4

Toxicity of Dieldrin to Various Fishes

Species	Exposure Time (hr)	Method	LC50 ug/l Dieldrin	Ref.
Bluegill (*Lepomis macrochirus*)	96	U	2.8	44
Juvenile White Mullet (*Mugil curema*)	48	F	7.1(TLM)	90
Shiner Perch (*Cymatogaster aggregata*)	96	F	1.50	93
Shiner Perch (*Cymatogaster aggregata*)	96	S	3.7	93
Dwarf Perch (*Micrometrus minimus*)	96	F	2.44	93
Dwarf Perch (*Micrometrus minimus*)	96	S	5.00	93
Atlantic Silverside (*Menidia menidia*)	24	S	10.	91
Atlantic Silverside (*Menidia menidia*)	96	S	5.	91
Striped Killifish (*Fundulus majalis*)	24	S	9.	91
Striped Killifish (*Fundulus majalis*)	96	S	4.	91
Bluehead (*Thalassoma bifasciatum*)	96	S	6.	91
Striped Mullet (*Mugil cephalus*)	96	S	23.	91
American eel (*Anguilla rostrata*)	48	S	4.	91
American eel (*Anguilla rostrata*)	96	S	.9	91
Mummichog (*Fundulus heteroclitus*)	24	S	20.	91
Mummichog (*Fundulus heteroclitus*)	96	S	5.	91
Northern Puffer(*Sphaeroides maculatus*)	96	S	34.	91
Striped Bass (*Morone saxatilis*)	96	F	19.7(TL50)	92

S=Static Bioassay
F=Flow-through Bioassay
U=Unknown

Data on the toxicity of aldrin and dieldrin ingested by aquatic organisms indicate that the compounds can be toxic at water concentrations of 2.03 ug/l and 1.5 ug/l respectively (93). Aldrin/dieldrin also has been reported to alter biological mechanisms of fish. Dieldrin at 0.36, 1.08, 3.6, and 10.8 ug in food fed to rainbow trout for 240 days *Salmo gairdneri*, altered brain concentrations of amino acids associated with ammonia detoxifying mechanisms, aspartate, glutamate and alanine, as well as the enzymes related to their metabolism (39).

The 24-hour TL50 (LC50) for rainbow trout, *S. gairdneri*, exposed to aldrin has been shown to be temperature dependent and to increases with increasing water temperature. At temperatures of 1.6 C, 7.2 C and 12.7 C the 24-hour TL50 (LC50) values were 24 ug/l, 8.1 ug/l, and 5.8 ug/l, respectively. Similarly, data for bluegills, *Lepomis macrochirus*, showed an effect of temperature. At temperatures of 12.7 C, 18.3 and C and 23.8C, the 24-hour LC50 concentrations were 36 ug/l, 16 ug/l and 10 ug/l, respectively (43). Data presented in Tables 5 and 6 illustrate that the 24-hour, 48-hour, and 96-hour TL50 (LC50) values are time and temperature related.

Dieldrin has been shown to affect adversely the ability of the freshwater fish, *Etheostoma nigrum*, to withstand thermal stress when exposed to a level of 2.3 ug/l for 30 days (41). The mortality of those exposed to aldrin was greater than the non-exposed population. In additon, changes in the oxygen consumption, whole body lipid and liver condition were affected adversely in fish after 15 days of exposure. These conditions later returned to within normal ranges except for liver damage which progressed with time.

Table 5

Effects of increasing temperature and exposure time on the toxicity of aldrin to bluegill* (44)

Temperature F	(C)	LC50 (ug/l) 24-hr.	48-hr.	96 hr.
45	(7.0)	130.	26.4	9.7
55	(12.7)	36.8	12.5	7.7
65	(18.3)	16.4	8.3	6.2
75	(23.8)	9.3	6.7	5.6

*weight of fish approximately 1 g.

Table 6

Effects of increasing temperature and exposure time on the toxicity of dieldrin to bluegill* (44)

Temperature F	C	LC50 (ug/l) 24-hr.	48-hr.	96-hr.
45	(7.0)	54	34	16
55	(12.7)	40	26	18
65	(18.3)	24	18	14.5
75	(23.8)	14	11	9.3
85	(29.4)	10	8.4	7.1

*weight of fish approximately 1 g.

d) Birds

The dieldrin oral LD50 for the sharp-tailed grouse is reported to be 6.9 mg/kg of body weight (94). Long-term feeding studies of birds have resulted in the characterization of a variety of sub-acute and chronic toxic effects attributable to aldrin and dieldrin, which are typical of the chlorinated hydrocarbons. Fertility among surviving female pheasants was lower than in the control group (45). Feeding dieldrin to pheasants at 6 mg/ per week for 13 weeks resulted in no mortality in the parents but the offspring of these hens when fed a diet with 6 mg dieldrin/week for 14 weeks showed 75 percent mortality. Visual cliff tests also showed adverse behavioral changes in chicks fledged from hens fed 8 mg/week for 14 weeks (45). Pen-reared, 5 week old pheasants were fed with encapsulated aldrin at 0.5, 1.0, and 1.5 mg/week for seven weeks, and another group with 0.5 mg on alternate days to a total dose of 1.5 mg. Treatments of birds between the ages of 5 and 21 weeks with either 1.0 mg/week or 1.5 mg total dose was found to depress growth. Fifty percent of the birds receiving the 1.5 mg/week dose died within 48 hours of the first treatment (46).

Dieldrin fed to Japanese quail at dietary levels of 0-40 ppm showed a definite relationship between dosage and mortality. The highest no-effect level as measured by growth, health or behavior was 10 ppm. At higher doses, egg production and fertility were reduced. Hatched chickes fed dietary levels greater than 10 ppm suffered mortalitites within 2 or 3 days of hatching (48).

The question of chlorinated hydrocarbons and their effects on avian calcium metabolism, steroid hormone metabolism and reproduction has been the subject of numerous investigations which have often resulted in conflicting data. The major cause of declining predatory bird populations in the last 20 years has been from a drastic drop in reproduction and not in the killing of adult birds. The failures in reproduction follow a similar pattern among the various species and involve delayed breeding or failure to lay eggs, thinning of eggshells and subsequent breakage, and high mortality in embroys and newly hatched birds. Recent studies show persistent organochlorine pesticides induce liver enzymes that lower estrogen levels and result in late breeding and other related reproductive manifestations (49).

Dieldrin was fed to 43 of 78 nesting female prairie falcons by tethering dieldrin-contaminated starlings (fed 10 ppm for 14 days) in sight of the falcons. Birds fed more than three treated starlings averaged dieldrin tissue levels eight times those of untested falcons. A straightline correlation was found between the amount of dieldrin consumed and the residual levels in the birds' fat and eggs. Eggshells from 34 untreated birds (egg dieldrin 1.9 ppm) were significantly thicker than from seven treated birds in which egg dieldrin averaged 41.5 ppm. At dieldrin concentrations of less than 20 ppm in the egg, there was no difference in the thickness of eggshells. The data establish a correlation between pesticide residues, thin eggshells, and poor hatching success (50). Studies of male chickens, pheasants, and quail exposed to aldrin showed the chemical to have a feminizing effect on all three. This is thought to be due to reduced testicular size and altered hormone metabolism (52). Reproduction of mallard ducks, pheasants, and bobwhite quail was reported reduced or inhibited by diets containing as little as 0.5 ppm aldrin (53).

e) Mammals

The LD50 for rats has been reported to be 54 to 56 mg/kg body weight for aldrin and 50 and 55 mg/kg for dieldrin (61). The oral dieldrin LD50 value for the dog is reported to be 65 to 95 mg/kg (61).

Administration of single oral dose of dieldrin to rats at 30 mg/kg of body weight resulted in impaired liver function (62). Impaired liver function also was found to occur in a number of animals species including man (62). Impairment of reprodution in a variety of mammalian species has been found to result from exposure to aldrin-dieldrin. A dieldrin dietary level of 100 ppm was found to induce abortion in guinea pigs and was lethal to one-third of the pregnant and one-half of the non-pregnant animals (64).

Dieldrin has been reported to be transferred from the mother to blastocyst and from mother to fetus in pregnant rabbits (54). In continuous feeding studies, neo-natal mortality in dogs and rats has been shown to increase (55, 56). A level of 2.5 mg/kg of 85 percent dieldrin has been reported to produce fetal malformations in Wistar rats (57).

Repetitive oral doses of up to 15 mg/kg dieldrin administered to pregnant sows during the last 30 days of gestation resulted in placental transfer to the embroys. Some degeneration of the kidneys tubules and slight hepatic lipidosis were osberved in the sows. No lesions were detected in the fetuses and there were no letal deaths or abortions (58).

Aldrin/dieldrin has also been shown to cause cytogenetic aberration in mice (59). In two studies comprising five long-term oral studies feeding dieldrin to CF-1 mice at various concentrations, liver enlargements and tumors were detectable. Appearance of tumors was dose responsive since tumors occurred 9 months following feed treatment with 10 ppm; 19 months with 5 ppm; and 23 months with 2.5 ppm. Further, the total groups all experienced a decrease in survival rates. At intake rates of 1.25 ppm and 1 ppm dieldrin, no liver enlargements were detected clinically and survival was not affected (60).

f) Human Health Hazard

Aldrin and dieldrin are highly mobile and persistent chemicals that are not lost by dilution or degradation in the inorganic components of the environment. The pesticides persist in the soil for several years, where they are absorbed by the roots and transported to the aerial parts of sequentially planted crops, such as soybeans and corn. Many of these products are important feed components for animals. The pesticide residues are thus incorporated, directly and indirectly, into the milk, meat, poultry, and soy products consumed by humans (See Appendices A and B hereto for an extensive discussion of the human hazards).

Evidence that aldrin-dieldrin poses a cancer hazard to man is provided by the mouse laboratory data. The carcinogencity of aldrin/dieldrin to mouse strains other than the CF-1 studies mentioned above (60) have been published (66). Although the liver is the principal organ affected in the mouse and was a major site of action in rats, there was also an increase in tumors of the lungs and other organs (66).

Aldrin poisoning of humans may occur by ingestion, inhalation and/or skin absorption. Severe symptons may result from ingestion or percutaneous absorption of as little as 1 gram, especially in the presence of liver disease. Renal damage, tremors ataxia, convulsions, followed by central nervous system depression, respiratory failure and death are symptoms resulting from acute exposure. Chronic exposures over a prolonged period may result in liver damage (66). In humans, pregnancy has been observed to offer a degree of protection from dieldrin intoxication to the mother, but at the expense of the fetus which concentrates dieldrin in its tissues (65).

Chemicals known to cause cancer in man have been identified only through epidemiological studies, either in the general public or in occupationally exposed workers. In the case of aldrin/dieldrin, epidemiological studies have not been possible because there are no clear-cut differential levels of exposure and because the period of exposure has been too short. Some cancers in man do not develop until late in life, usually 20 years or more after initial exposure (66). Animal studies are accepted as determining factors when assessing the carcinogenic potential of a chemical to man.

Mouse and rat systems are commonly accepted experimental animal species, both because their relatively short life span permits lifetime testing within a reasonable period of time and because the pathological development of tumors in these species is particularly well known and understood (66). A number of experiments have shown, as noted above, that aldrin/dieldrin induces cancer in five different strains of mice and perhaps in the rat. Based on these data this Agency concluded in the FIFRA proceedings, noted above, that aldrin/dieldrin pose a serious cancer risk and health threat to man (66).

III. ENVIRONMENTAL FATE AND EFFECTS

Movement of aldrin and dieldrin into the aquatic ecosystem is of critical importance since, once having entered water, these chemicals are extremely persistent and toxic. Basically, as with other organochlorine pesticides, aldrin/dieldrin enters water by one of three routes, physical transport, chemical transport or biolgical transport. It is virtually impossible to identify all of the various physical factors affecting movement of persistent organic chemicals such as aldrin/dieldrin(70)

Characteristics of the soil in which aldrin and dieldrin are found are of importance in determining the rate of movement of the pesticide. It has been found that volatilization is one means of loss of these pesticides from sand and moist soils with low organic content. Temperature is another parameter of considerable importance, as it has been found that the half-life of dieldrin in a sandy loam decreased with temperature; however, this loss apparently is not due to volatilization (71).

Aldrin and dieldrin are persistent in the environment, but aldrin is readily converted to dieldrin in both living and non-living sites (72). After its conversion from aldrin, dieldrin is metabolized or degraded under a variety of circumstances, but generally at a very slow rate. Some microorganisms, insects and mammals have been shown to degrade dieldrin and under certain conditions. Sunlight can cause similar degradation. Overall decomposition rates, however, are inadequate to prevent its persistence in the biosphere (14). The photoconversion of aldrin by ultraviolet light should be emphasized, since in nature residues of this pesticide in sunlight could result in reactions significantly affecting living organisms. Experimental UV irradiation of aldrin has been shown to produce dieldrin and aldrin photoconversion isomers almost quantitatively (73, 74). There is evidence that these isomers are even more toxic than the original compounds when tested with insects and fish (75). In general, although aldrin and dieldrin are not highly reactive chemically in the environment, the reactions they undergo tend to increase their potential for harmful biological effects (23).

A fundamental fact to be emphasized in considering organochlorine pesticides in the biosphere is their virtual water insolubility and high lipid solubility, which facilitates storage by fatty tissues. Aldrin and dieldrin are preferentially soluble in living (especially lipid) systems which almost always indicate slower metabolism and turnover than observed in aqueous interactions (14, 16). Microscopic plants and animals present highly variable responses to aldrin and dieldrin. Although almost all of them accumulate these chemicals to some extent, it is probable that they are relatively immune to acute lethal effects. In algae, aldrin and dieldrin do not build up to levels seen with DDT; however, 16,000-fold magnification over the concentration in the medium has been observed (76). Over 500 bacterial isolates have been studied from soil and few of them have been found to degrade dieldrin (9). In this study, Matsumura and Boush concluded:

(1) Dieldrin is one of the most stable and hazardous insecticides in our environment.

(2) This persistence suggests it is of low biochemical reactivity .

(3) The potent effects of aldrin/dieldrin depend on forming physical complexes with nervous systems of insects and mammals.

(4) Most chlorinated hydrocarbon insecticides have little effect on bacterial and fungal growth.

(5) Many microbial changes brought about by application of these insecticides to soil may be attributable to secondary effects.

As noted above, the extent of lipid metabolism probably is a decisive parameter in bioaccumulation of these substances as evidenced by laboratory studies which show concentration factors for dieldrin from water of 114,935 in snails, 7,480 in algae, 6,145 in fish, 247 in crabs and 1,015 in clam (16).

Parrish (34) reported whole body residues in spot (*Leiostomus xanthurus*) as much as 6,000 times water concentration in 11 to 18 days' exposure.

The alga, *Scenedesmus obliguus*, the waterflea, *Daphnia magna*, and the guppy, *Poecilia reticulata*, have been found to accumulate dieldrin directly from water. The average concentration factors (concentration in organisms, dry weight, divided by concentrations in water) were 1,282 for algae, 13,954 for *D. magna*, and 40,307 (estimated) for the guppy (79). The amount accumulated by each species at equilibrium was directly proportional to the concentration of dieldrin in water. Accumulation of dieldrin by guppies resulted from exposure to either contaminated water or to their food source, which was *Daphnia magna* (79). The ostracod, *Chlamydotheca arcuata*, has been shown to accumulate dieldrin at levels of 12,000 to 260,000 times that of the initial theoretical concentration in water (77).

Studies on degradation of dieldrin by biological systems other than microbial have been also relatively unsuccessful. Because of these findings it has been concluded that no biological systems are important in reducing the actual toxicity of dieldrin entering their metabolism (9).

Residues of aldrin and dieldrin have been found in most molluscs, fishes, birds and mammals studied regardless of location in the world (19, 20). Rainwater, drinking water, and non-potable waters in Hawaii were sampled and found to contain dieldrin in the low parts per trillion range (78).

Aldrin-dieldrin intake in mammalian systems has been shown to cause liver damage, kidney damage, and behavioral disturbances (10, 58, 60, 62, 80).

Data from laboratory studies using mice have demonstrated that dieldrin is a potent carcinogen (60, 10) and potential carcinogenic danger to humans experiencing intake of low dieldrin levels from either food or water has been established (66).

IV. CRITERIA FORMULATION

The persistence, bioaccumulation potential and carcinogenicity of aldrin-dieldrin make avoidable human exposure unreasonably hazardous. A chronic criterion .003 ug/l would provide for the protection of aquatic life.

Aldrin-Dieldrin has been found to be toxic to aquatic organisms at low levels. The Aldrin-Dieldrin 96-hr LC50 to fish is reported as low as 1.50 ug/l for shiner perch, *Cymatogaster aggregata* (93). The 96-hr LC50 for striped mullet, *Mugil cephalus* is 23 ug /l, (91), and for the striped bass, *Morone saxatilis* the 96-hr TL50 (LC50) has been shown to be 19.7ug/l (92).

Residue accumulation of dieldrin and aldrin is well documented. Levels of dieldrin in fish tissue from Lake Michigan have been as much as 100,000 times the dieldrin levels occurring in the water (68).

Laboratory exposures of fish, invertebrates, and algae have indicated that residue accumulation of aldrin and dieldrin is significant. The reticulate sculpin (*Cottus perplexus*) exposed to 0.017 ug/l dieldrin in water for 32 days developed tissue concentrations of 50,000 times the water exposure level (82). The sailfin molly (*Poecilia latipinna*) exposed for 34 weeks to 12, 6, 3, 1.5 and 0.75 ug/l dieldrin in water concentrated dieldrin in all tissues at least 10,000 times (35). At the termination of a 64-week exposure of the Ostracod (*Chlamydotheca arcuata*) to water concentrations of aldrin at 0.01 and 0.10 ug/l and dieldrin at 0.01 and .10 ug/l, dieldrin recovered from the tissue (dry weight basis) were 12,000 to 260,000 times the initial theoretical water concentrations (77). In a model ecosystem study, residue accumulation factors for dieldrin were determined to be 114,235 times water concentration for the snail, 7,480 times water concentration for algae, 6,145 times water concentration for fish, 2,145 times water concentration waterfleas, *Daphnia, sp.*, 1,280 times water concentration for a pond weed, *Elodea*, and 247 times water concentration for the crab, and 1,015 times water concentration for the clam (16). Other bioaccumulation studies have indicated similar uptake levels (76, 79, 83, 84). With dieldrin at a concentration of 0.5 ug/l, the rate of uptake by the crab larvae (*Leptodius floridanus*) in 18 days was 0.191 ug/g per day from water (85).

In long-term feeding studies, 1 mg/kg dieldrin affected reproduction in the Hungarian partridge (88). Slight eggshell thinning was noted in mallard ducks fed a diet containing 1.6 ppm (mg/kg) of dieldrin (87). Deer were affected adversely by long-term feeding of a diet containing 5 ppm (mg/kg) of dieldrin (88).

The highly mobile and persistent nature of aldrin-dieldrin, as well as its capability for becoming incorporated into products consumed by humans, results in an imminent human health hazard in view of its carcinogenicity. Walker, et al. (50) demonstrated the tumorigenic activity of dieldrin to CF-1 mice. Levels which resulted in tumor formation can be found in aquatic food chain organisms as a result of bioaccumulation from water.

Bioaccumulation studies with aldrin-dieldrin have shown that tissue residues up to 100,000 times the ambient water concentration occur in fish (68). Since the FDA tissue residue guideline for aldrin/dieldrin is 0.3 ug/l, water levels higher than.003 ug/l could result in bioaccumulation to levels above .3 ppm in fish flesh. Therefore the chronic criterion for aquatic life is set at.003 ug/l. The primary impact of this bioaccumulation in fish and their food sources centers on the biological transport of aldrin-dieldrin to birds and mammals including man. The chronic toxicity criterion of 0.003 ug/l is based on toxicity factors apart from carcinogenicity.

References

1. Bann, J. M., T. J. DeCino, N. W. Earle and Y. P. Sun. 1956. The fate of aldrin and dieldrin in the animal body. Jour. Agri. Food Chem. 4:937.

2. Brooks, G. T. 1960. Mechanism of resistance of the adult housefly (Musca domestica) to, cyclodiene, insecticides. Nature. 186:96.

3. Cohen, A. J. and J. N. Smith. 1961. Fate of aldrin and dieldrin in locusts. Nature. 189:600.

4. Lichtenstein, E. P. and K. R. Schulz. 1960. Epoxidation of aldrin and heptachlor in soils as influenced by autoclaving, moisture, and soil types. Jour. Econ. Entom. 53:192.

5. Krieger, R. I., P. P. Feeny, and C. F. Wilkinson. 1971. Detoxication enzymes in the guts of caterpillars: An evolutionary answer to plant defenses? Science. 172:579.

6. Ludke, J. L., J. R. Gibson and C. I. Lusk. 1972. Mixed function oxidase activity in freshwater fishes: Aldrin epoxidation and parathion activation. Toxic. Appl. Pharm. 21:89.

7. Khan, M. A. Q., A. Kamal, R. J. Wolin and J. Runnels. 1972. *In vivo* and *In vitro* epoxidation of aldrin by aquatic food chain organisms. Bull. Environ. Contam. and Toxic. 8:219.

8. Carlson, G. P. 1974. Epoxidation of aldrin to dieldrin by lobsters. Bull. Environ. Contam. and Toxic. 11:577.

9. Matsumura, F. and G. M. Boush. 1967. Dieldrin: Degradation by soil microorganisms. Science. 156:959.

10. IARC Working Group. 1974 Dieldrin. IARC monographs on the Evaluation of Carcinogenic Risk of Chemicals to man: Some organochlorine pesticides. 5:125.

11. Chacko. C. I., J. L. Lockwood and M. Zabik. 1966. Chlorinated hydrocarbon pesticides: Degradation by microbes. Science. 154:893.

12. Edwards, C. A. 1966. Insecticide residues in soils. Residue Reviews. 13:83.

13. Wedemeyer, G. 1968. Partial hydrolysis of dieldrin by *Aerobactor aerogenes*. Applied Microbiology. 16:661.

14. Westlake, W. E. and J. P. San Antonio. 1960. Insecticide residues in plants, animals and soils. *In*: The nature and fate of chemicals applied to soils, plants, and animals. U. S. Dept. of Agriculture, ARS 20. 9:105.

15. Nash, R.G. and E.A. Woolson. 1967. Persistence of chlorinated hydrocarbon insecticides in soils. Science. 157:924.

16. Sanborn, J.R. and C.C. Yu. 1973. The fate of dieldrin in a model ecosystem. Bull. Environ. Contam. and Toxic. 10: 340.

17. Brockway, D.L. 1973. The uptake, storage and release of dieldrin and some effects of its release in the fish, *Cichlosoma bimaculatum* (Linnaeus). Diss. Abst. International. 33:4323B.

18. Grzenda, A.R., W.J. Taylor, and D.F. Paris. 1972. The elimination and turnover of 14C-dieldrin by different goldfish tissues. Tran Amer. Fish. Soc. 101:636.

19. Butler, P.A. 1973. Organochlorine residues in estuarine mollusks. National Pesticide Monitoring Program. Pest. Monit. Jour. 6:232.

20. Wurster, C.F. 1971. Aldrin and dieldrin. Environment. 13:23.

21. Jones, L.W. 1956. Effects of some pesticides on microbial activitie of the soil. Utah State Agric. College Bull. 390:1.

22. Cairns, Jr., J. 1968. The effects of dieldrin on diatoms. Mosquito News. 28:177.

23. Gaur, A.C. and M.C. Bardiya. 1970. The effect of lindane and dieldr on soil microbial populations. Indian Jour. Microbiology. 10:33.

24. Harrington, R.W. and W.L. Bidlingmayer. 1958. Effects of dieldrin on fishes and invertebrates of a salt marsh. Jour. Wildl. Mgmt. 22:76.

27. Sanders, H.O. and O.B. Cope. 1968. The relative toxicities of several pesticides to naiads of three species of stoneflies. Limnol. Oceanog. 13:112.

28. Eisler, R. 1969. Acute toxicities of insecticides to marine decapod crustaceans. Crustaceana. 16:302.

29. Sanders, H.O. 1969. Toxicity of pesticides to the crustacean. *Gamm lacustris*. Tech. Paper 25, Bur. Sport Fish. Wildl., U.S. Depart of the Interior, 18p.

30. Federal Water Pollution Control Administration. 1968. Water quality criteria. Report of the National Tech. Advis. Comm. to Sec. of the Interior. Fed. Water Poll. Cont. Admin., U.S. Department of the Interior, 234 p.

31. Sanders, H.O. and O.B. Cope. 1966. Toxicities of several pesticides to two species of Cladocerans. Trans. Amer. Fish Soc. 95:165.

32. Menzel, D.W., J. Anderson and A. Randtke. 1970. Marine phytoplankton vary in their response to chlorinated hydrocarbons. Science :1724.

34. Parrish, P.R., J.A. Couch, J. Forester, J.M. Patrick, and G.H. Cook. 1973. Dieldrin: Effects on several estuarine organisms. Contrib. No. 178, Gulf Breeze Environ. Res. Lab. Proceedings of the 27th Annual Conference of the Southeastern Association of Game and Fish Commissioners.

35. Lane, C.E. and R.J. Livingston. 1970. Some acute and chronic effects of dieldrin on the sailfin molly, *Poecilia latipinna*. Trans. Amer. Fish. Soc. 3;489

36. Cairns, J., Jr., N.R. Foster, and J.J. Loos. 1967. Effects of sublethal concentrations of dieldrin on laboratory populations of guppies, *Poecilia reticulata* Peters. Proc. Acad. Nat. Sci., Phila., 119:75.

37. Jensen, L.D. and A.R. Gaufin. 1966. Acute and long-term effects of organic insecticides on two species of stonefly naiads. Jour. Water Poll. Cont. Fed. 38;1273.

38. Epifanio, C.E. 1971. Effects of dieldrin in seawater on the development of two species of crab larvae, *Leptodius floridanus* and *Panopeus herbstii*. Mar. Biol. 11;355.

39. Mehrle, P.M. and R.A. Bloomfield. 1974. Ammonia detoxifying mechanisms of rainbow trout altered by dietary dieldrin. Toxic. Appl. Pharm. 27;355.

40. Henderson, C., G.H. Pickering and C.M. Tarzwell. 1959. The toxicity of organic phosphorus and chlorinated hydrocarbon insecticides to fish. Biol. Problems in Water Pollution, Trans. 1959 Seminar. Robert A. Taft San. Eng. Center, Tech. Rep W60-3;76.

41. Silbergeld, E.K., 1973. Dieldrin: Effects of chronic sublethal exposure on adaptation to thermal stress in freshwater fish. Environ. Sci. Tech., 7:846.

42. Chadwick, G. and D.L. Shumway. 1969. Effects of dieldrin on the growth and development of steelhead trout. In: The biological impact of pesticides in the environment. Environ. Health Sciences Series No. 1, Oregon State University, pp. 90-96.

43. Macek, K.J., Hutchinson, C. and Cope, O.B. 1969. The Effects of temperature on the susceptibility of bluegills and rainbow trout to selected pesticides. Full. Envir. Cont. and Tox. 4:174.

44. Cope, O.B. 1965. Sport Fishery Investigations. Fish and Wildl. Serv. Circ. 226, Bur. Sport. Fish Wildl. pp. 51-64.

45. Baxter, W.L., R.L. Linder and R.B. Dahlgren, 1969. Dieldrin effects in two generations of penned hen pheasants. Jour. Wildl. Mgmt. 33:96.

46. Hall, J.E., Y.A. Greichus and K.E. Severson. 1971. Effects of aldrin on young pen-reared pheasants. Jour. Wildl. Mgmt. 35:429.

48. Walker, A. I. T., C. H. Neill, D. E. Stevenson and J. Robinson. 1969. The toxicity of dieldrin (HEOD) to Japanese quail (Coturnix coturnix japonica) Toxicol. Appl. Pharmacol. 15:69.

49. Peakall, D. B. 1970. Pesticides and the reproduction of birds. Sci. Amer. 222:73.

50. Enderson, J. H. and D. D. Berger. 1970. Pesticides: Eggshell thinning and lowered production of young in prairie falcons. Bio. Sci. 20:355.

52. Lutz-Ostertag, Y. and H. Lutz. 1969. Preliminary note on the "estrogenic" effects of "aldrin" on the urogenital tract of the bird embryo. Compt. Rend.; Res. D.269(4): 484. Health Aspects of Pest. Abst. No. 70-0627.

53. Chichester, C. O. (editor). 1965. Research in pesticides. Academic Press, New York.

54. Hathaway, D. E., J. A. Moss, J. A. Rose and D. J. M. Williams. 1967. Transport of dieldrin from mother to blastocyst and from mother to foetus in pregnant rabbits. European Jour. Pharmacol. 1:167.

55. Treon, J. F. and F. P. Cleveland. 1955. Toxicity of certain chlorinated hydrocarbon insecticides for laboratory animals, with special reference to aldrin and dieldrin. Agric. Food Chem. 3:402.

56. Kitselman, C. H. 1953. Long term studies on dogs fed aldrin and dieldrin in sublethal dosages, with reference to the histopathological findings and reproduction. Jour. Amer. Vet. Med. Assn. 123:28.

57. Boucard, M., I. S. Beaulaton, R. Mestres, M. Allieu and S. Cabane. 1970. Teratogenesis: Effect of the period and duration of treatment. Therapie. 25:907.

58. Uzoukwu, M. and S. D. Sleight. 1972. Effects of dieldrin in pregnant sows. Jour. Amer. Vet. Med. Assn. 160:1641.

59. Markarjan, D. W. 1966. Cytogenetic effect of some chlororganic insecticides on the nuclei of mouse bone-marrow, cells. Geneticla. 2:132.

60. Walker, A. I. T., E. Thorpe and D. E. Stevenson. 1972. The toxicology of dieldrin (HEOD). I. Long-term oral toxicity studies in mice. Food Cosmet. Toxicol. 11:415.

61. Spector, W. S. (ed.) 955. Handbook of toxicology. Volume I: Acute Toxicities of Solids, Liquids and Cases to Laboratory Animals. NAS-NRC. W.B. Saunders Co., Philadelphia. pp. 14, 15, 96, 97.

62. Bhatia, S. C., S. C. Sharma and T. A. Venkitasubramaniam. 1972. Effect of dieldrin on certain enzyme systems of rat liver. Br. Jour. Exp. Pathol. 53:419.

64. Uzoukwu, M. and S. D. Sleight. 1972. Dieldrin toxicosis: Fetotoxicosis, tissue concentrations, and microscopic and ultrastructural changes in guinea pigs. Amer. Jour. Vet. Res. 33:579.

65. Nowak, W., W. Lotocki, H. Szrzedzinski, A. Strasiewicz and J. Badurski. 1971. Dieldrin poisoning during pregnancy. Pol. Tyg. Lek. 25:958. Health Aspects of Pest. Abst. No. 72-0532.

66. Shell Chemical Company, et al. Recommended decision of Administrative law Judge. EPA FIFRA Docket Nos. 145 etc. 39 F.R. 37246-37265 (October 18, 1975). Reprinted in Appendix A hereto.

68. Reinert, R. E. 1970. Pesticide concentrations in Great Lakes fish. Pest. Monit. Jour. 3:233.

70. Peterle, T. J. 1969. Translocation of pesticides in the environment. In: The biological impact of pesticides in the environment--a symposium assessing the significance of pesticides in relation to ecological problem health. Environmental Health Series No. 1. Proceedings of symposium held August 18-20, at Oregon State Univ., Corvallis, Oregon. P1-210.

71. White, A. W. 1970. The influence of moisture, temperature, and soil properties on disappearance of lindane and dieldrin from soil. Dissertation Abstracts International. 30:5327

73. Rosen, J. D. and D. J. Sutherland. 1967. The nature and toxicity of the photoconversion products of aldrin. Bull. Environ. Contam. and Toxicol. **2**:1.

74. Crosby, D. G. and K. W. Moilanen. 1974. Vapor-phase photodecomposition of aldrin and dieldrin. Arch. Environ. Contam. and toxicol. **2**:62.

75. Georgacakis, E., S. R. Chandran, and M. A. Q. Khan. 1971. Toxicity-metabolism relationship of the photoisomers of cyclodiene insecticides in freshwater animals. Bull. Environ. Contam. Toxicol. **6**:535.

76. Rice, C. P. and H. C. Sikka. 1973. Fate of dieldrin in selected species of marine algae. Bull. Environ. Contam. Toxicol. **9**:116.

77. Kawatski, J. A. and J. C. Schmulbach. 1971. Accumulation of insecticide in freshwater ostracods exposed continuously to sublethal concentrations of aldrin or dieldrin. Trans. Amer. Fish. Soc. :565.

78. Bevenue, A., J. W. Hylin, Y. Kawano, and T. W. Kelley. 1972. Organochlorine residues in water, sediment, algae and fish, Hawaii--1970-1971. Pest. Monit. Jour. **6**:56.

79. Reinert, R. E. 1972. Accumulation of dieldrin in an alga (*Scenedesmus obliqus*), *Daphnia magna* and the guppy (*Poecilia reticulata*). Jour. Fish. Res. Bd. Can. **29**:1413.

80. Avar, P. and G. Czegledi-Janko. 1970. Occupational exposure to aldrin: Clinical and laboratory findings. Brit. Jour. Ind. Med. **27**:279.

82. Chadwick, G. G. and R. W. Brocksen. 1969. Accumulation of dieldrin by fish and selected fish-food organisms. Jour. Wildl. Mgmt. **33**:693.

83. Johnson, B. T., C. R. Saunders and H. O. Sanders. 1971. Biological magnification and degradation of DDT and aldrin by freshwater invertebrates. Jour. Fish. Res. Bd. Can. **28**:705.

84. Petrocelli, S. R., A. R. Hanks, and J. W. Anderson. 1973. Uptake and accumulation of an organochlorine insecticide (dieldrin) by an estuarine mollusc *Rangia cuneata*. Bull. Environ. Contam. Toxicol. **10**:315.

85. Epifanio, C. E. 1973. Dieldrin uptake by larvae of the crab *Leptodius floridanus*. Marine Biology. **19**:320.

86. Neill, D. D., *et al.* 1969. 58th Annual Meeting Poul. Sci. Assn. 76.

87. Lehner, P. N. and A. Egbert. 1969. Dieldrin and eggshell thickness in ducks. Nature. *224*:1218.

88. Murphy, D. A. and L. J. Korschgen. 1970. Reproduction, growth, and tissue residues of deer fed dieldrin. Jour. Wildlife Mgmt. *34*:887.

89. Patil, K.C., F. Matsumura, and G.M. Boush. 1972. Metabolic transformation of DDT, dieldrin, aldrin, and endrin by marine microorganisms. Environ. Sci. and Tech. *6*:631.

90. Butler, Philip A.. Commercial fisheries investigation. In: Pesticide Wildlife Studies: A Review of Fish and Wildlife Service Investigations During 1961 and 1962, U.S. Dept Interior, Fish and Wildlife Service Circular 167, pp 11-25.

91. Eisler, R. 1970. Acute toxicities of organochlorine and organophosphorus insecticides to estuarine fishes, In: Technical Papers of the Bureau of Sport Fisheries and Wildlife, No.46, U.S. Government Printing Office, Wash. D.C.

92. Korn, S., and R. Earnest, 1974, Acute toxicity of twenty insecticides to striped bass, *Morone saxatilis*, Calif. Fish and Game 60: 128.

93. Earnest, R., and P. E. Benville, Jr. 1972, Acute toxicity of four organochlorine insecticides to two species of surf perch, California Fish and Game. 58: 127.

94. McEwen, L. C., and R. L. Brown, 1966. Acute toxicity of dieldrin and malathion to wild sharp-tailed grouse, Jour. Wildlife Mgmt. 30:604.

GLOSSARY

Acutely toxic: Causing death or severe damage to an organism by poisoning during a brief exposure period, normally ninety-six hours or less.

Anadromous fishes: Fishes that spend a part of their lives in seas or lakes, but ascend rivers and streams at certain intervals to spawn. Examples are sturgeon, shad, salmon, trout, and striped bass.

Application factor: The ratio of the safe concentration to the lethal concentration as determined for potential aquatic pollutants administered to species of interest.

Bioaccumulation (Bioconcentration): The phenomenon wherein elements or compounds are stored in living organisms because elimination fails to match intake.

Carcinogenic: Producing cancer.

Catadromous fishes: Fishes that feed and grow in fresh water, but return to the sea to spawn. The best example is the American eel.

Chronically toxic: Causing death or damage to an organism by poisoning during prolonged exposure, which, depending on the organism tested and the test conditions and purposes, may range from several days, to weeks, months, or years, or through a reproductive cycle.

EC50: The concentration at which a specified effect is observed under the test conditions in a specified time in fifty percent of the organisms tested. Examples of specified effects are hemorrhaging, decreased feeding, dilation of pupils, and altered swimming patterns.

Epilimnion: That region of a body of water that extends from the surface to the top of the thermocline and does not have a permanent temperature stratification.

Flow-through bioassay: An assay system in which aquatic species are exposed to toxicants in a constantly flowing system, and where the toxicant is replenished continuously or discontinuously.

Hardness (Water): The concentration of the polyvalent metallic ions dissolved in water. Usually it is reported as the equivalent concentration of calcium carbonate ($CaCO_3$).

Hyperplasia: Abnormal multiplication or increase in the number of normal cells in normal arrangement in a tissue.

Hypolimnion: The region of a body of water that extends from the bottom of the thermocline to the bottom of the water body and is essentially independent of most surface phenomena.

LC25: The concentration of a toxicant that is lethal (fatal) to twenty-five percent of the organisms tested under the test conditions in a specified time.

LC50: The concentration of a toxicant which is lethal (fatal) to fifty percent of the organisms tested under the test conditions in a specified time. It is virtually identical with TLm and TL50.

LD50: The dose of a toxicant that is lethal (fatal) to fifty percent of the organisms tested under the test conditions in a specified time. A dose is the quantity actually administered to the organism and is not identical with a concentration, which is the amount of toxicant in a unit of test medium rather than the amount ingested by or administered to the organism.

Liter (l): The volume occupied by one kilogram of water at a pressure of 760 mm of mercury and a temperature of 4^{o} C. A liter is 1.057 quart.

Methylmercury: Mercury which has been methylated, usually through some biological agent, such as bacteria.

Microgram per liter (ug/l): The concentration at which one millionth of a gram (one microgram) is contained in a volume of one liter. Where the density of solvent is equal to one, one ug/l is equivalent to one part per billion (ppb) or one microgram per kilogram (ug/kg).

Microgram per kilogram (ug/kg): The concentration at which one millionth of a gram (one microgram) is contained in a mass of one kilogram. A kilogram is 2.2046 pounds.

Milligram per kilogram (mg/kg): The concentration at which one thousandth of a gram (one milligram) is contained in a mass of one kilogram. A gram contains 1000 milligrams.

Milligram per liter (mg/l): The concentration at which one milligram is contained in a volume of one liter. Where the density of the solvent is equal to one, one mg/l is equivalent to one part per million (ppm) or one milligram per kilogram (mg/kg).

Milliliter (ml): A volume equal to one thousandth of a liter.

Nanogram per liter (ng/l): The concentration at which one billionth of a gram (one nanogram) is contained in a volume of one liter. Where the density of the solvent is equal to one, one ng/l is equivalent to one part per trillion or one nanogram per kilogram (ng/kg).

Neoplastic: Describing any new and abnormal growth, such as a tumor.

Part per million (ppm): A concentration in which one unit is contained in a total of a million units. Any units may be used (e.g., weight, volume) but in any given application identical units must be used (e.g., grams per million grams or liters per million liters). Where the density of the solvent is one, one part per million is equivalent to one milligram per liter.

Parts per thousand (o/oo): A concentration at which one unit is contained in a total of a thousand units. The rules for using this term are the same as those for parts per million. Normally, this term is used to specify the salinity of estuarine or sea waters.

Piscicide: A substance used for killing fish.

Static bioassay: A bioassay in which the toxicant is not renewed during the test.

Thermocline: That layer in a body of water where the temperature difference is greatest per unit of depth. It is the layer in which the drop in temperature is 1 °C. or greater per meter of depth.

TLm - Median Tolerance Limit: The concentration of a test material at which fifty percent of the test animals are able to survive under test conditions for a specified period of exposure. It is virtually synonymous with LC50 and TL50.

TL50: Synonymous with TLm and virtually synonymous with LC50.

Tumorigenic: Causing or producing tumors.

Appendix A

Reproduced from best available copy.

[FRL 278-7; FIFRA Docket Nos. 145 etc.]

SHELL CHEMICAL CO. ET AL.

Consolidated Aldrin/Dieldrin Hearing

On August 2, 1974, I issued my Notice of Intent to Suspend the registrations of certain pesticide products containing Aldrin and Dieldrin. After an adjudicatory hearing, the Chief Administrative Law Judge of this Agency on September 20, 1974, issued a recommended decision concerning the allegations contained in that Notice of Intent to Suspend. On October 1, 1974, I issued my Opinion and Order. The three documents are published herewith.

Dated: October 8, 1974.

RUSSELL E. TRAIN,
Administrator.

[F.I.F.R.A. Dockets Nos. 145, etc.]

SHELL CHEMICAL CO. ET AL.

NOTICE OF INTENTION TO SUSPEND AND FINDINGS AS TO AN IMMINENT HAZARD

In the matter of Shell Chemical Company, et al., Registrants (Consolidated Aldrin/Dieldrin Hearing) F.I.F.R.A. Dockets Nos. 145 etc.

By this order, issued pursuant to section 6(c) of the Federal Insecticide, Fungicide, and Rodenticide Act, as amended ("FIFRA"), I am hereby serving notice of my intent to suspend the registrations and prohibit the production for use of all pesticide products containing Aldrin or Dieldrin which are subject to and for which appeals were duly filed from the Aldrin/Dieldrin cancellation order issued by the Administrator of the Environmental Protection Agency on June 26, 1972.[1] This suspension order is effective within five days unless the registrants request an expedited hearing pursuant to section 6(c)(2), with the added provision that I am permitting, pursuant to section 15(b)(2), use or sale of existing formulated stocks of pesticides containing Aldrin or Dieldrin which are on hand as of the effective date of the suspension order. Such hearing, if requested, shall take no longer than 15 days from the commencement of the hearing, unless, for good cause shown I extend that time for no more than 5 additional days

Background. The history of prior attempts to regulate the sale and use of Aldrin and

---

[1] In the matter of Shell Co., et al., I.F.&R. Docket No. 145 etc., F.R., Vol. 39, No. 126, at p. 12904 (published June 29, 1972). For purposes of clarification, the result of a final order of suspension will be to prohibit the manufacture of Aldrin or Dieldrin for any use except for the three uses permitted by the June 26, 1972, order. Those three exempted uses are: Restricted termite use, the dipping of roots and tops of non-food plants and use in a total effluent-free mothproofing system.

Dieldrin is both lengthy and involved. The original petition for the cancellation and immediate suspension of all uses of Aldrin and Dieldrin was filed by the Environmental Defense Fund (EDF) on December 3, 1970. Shortly thereafter, on March 18, 1971, the Administrator of EPA announced the issuance of appropriate notices of cancellation based on a finding of "a substantial question as to the safety" of Aldrin and Dieldrin. At the same time the Administrator concluded that current uses of the compounds did not pose "an imminent hazard to the public," as that standard was interpreted in that Order, and he thus refrained from ordering a suspension of the compounds pending completion of the administrative procedure of review provided by the governing statute, the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA), 7 U.S.C. §§ 135 et seq., since amended by Pub. L. 92–516, 86 Stat. 973, October 21, 1972 (FIFRA-amended). The Administrator's failure to suspend the registrations prompted the filing by EDF of a petition for review in the U.S. Court of Appeals for the District of Columbia. A decision was issued by the Court of Appeals on May 5, 1972. "Environmental Defense Fund v. Environmental Protection Agency," 465 F.2d. 528 (1972). In that decision the Court remanded the record to EPA for further reconsideration of the issue of suspension, in light of the judicial interpretation of the power of suspension enunciated in the decision and the recently released Aldrin/Dieldrin scientific Advisory committee report.' The Court specifically called upon EPA to explicate the nature and extent of evidence available on the carcinogenicity of Aldrin/Dieldrin.

On June 26, 1972, upon review of the scientific advisory committee report and all other available data the Administrator reaffirmed the cancellation of nearly all Aldrin/Dieldrin products. In addition, that same order solicited views from the general public on the question of whether any of the cancelled uses should also be suspended. Particular emphasis was given to those methods of application and formulation (i.e., aerial application and dust formulation) presenting the most obvious risk of widespread unavoidable dissemination of the compounds.

On December 7, 1972, the Administrator announced that immediate curtailment of all aerial applications, dust formulations and use of these products for fire ant control and in moth proofing systems involving effluent discharge had been achieved through the voluntary cooperation of those affected Aldrin and Dieldrin registrants. The Administrator, in response to the Order of the Court of Appeals, again re-examined the issue of suspension. Based on a review of the evidence available at that time the Administrator again declined to exercise his power of suspension pending the completion of the hearing. His decision was based on the belief that the current uses did not present a substantial likelihood that serious harm would be experienced during the 12–18 months in which the hearing was expected to be completed.

II *Basis for current re-evaluation of suspension*. In the initial Aldrin/Dieldrin cancellation Order, former Administrator William D. Ruckelshaus stated then that the Agency would be prepared to re-evaluate the question of suspension at any later stage in the administrative proceedings (March 18th Statement, 1971, p. 12). In this Agency's brief to the U.S. Court of Appeals for the District of Columbia filed in response to the earlier EDF appeal on the Aldrin/Dieldrin suspension issue, EPA readily acknowledged that: "The concept of the safety of the product is an evolving one which is constantly being further refined in light of our increasing knowledge." Indeed, as the Court of Appeals emphasized in its opinion remanding the Aldrin/Dieldrin suspension issue to this Agency: "The administrative process is a continuing one, and calls for continuing reexamination at significant junctures." "Environmental Defense Fund, Inc. v. Environmental Protection Agency," supra, citing "American Airlines, Inc. v. C.A.B.", 359 F. 2d 624 (en banc), cert denied 385 U.S. 843 (1966).

There is no question but that the current proceeding involving the continued registration and future manufacture and use of Aldrin/Dieldrin products is at a "significant juncture." It has been estimated that the taking of evidence alone in this hearing will continue for another 4 or 5 months. This means that a final Agency decision cannot be expected until sometime in early 1975. Thus, the time period for a final decision as projected by the Administrator in December 1972 has grown considerably. Absent this Order there is nothing to prevent the manufacturer, during the period prior to a final decision on cancellation, from producing an additional estimated 10 million or more pounds of active technical product Aldrin for anticipated 1975 sales. This will mean that after formulation of the technical product over 50 million pounds of formulated final products will be available for sale and possible use over the period of the next year. The manufacturing process which would produce the Aldrin/Dieldrin products for sale and use in 1975 has been scheduled, according to the sole manufacturer, Shell Chemical Company, to begin on September 1, 1974. Shell has refused to delay voluntarily the manufacture of these products until completion of the current cancellation hearing. If after the end of the cancellation proceeding I decide finally to prohibit the use of these pesticides and yet the current manufacturing cycle is permitted to be completed, the disposal of such tremendous amounts of these chemicals will present enormous environmental risks and problems, discussed further below, which must be anticipated and avoided by this action. Once the manufacturing process is completed such risks are irrevocably created.

This proceeding is at a "significant juncture" in another highly significant sense in that an intense examination of the relevant evidence over the past year has brought to light certain previously unknown facts, which have now been reviewed and scientifically documented for the first time. On March 22, 1974, this Agency's Office of Hazardous Materials Control, through the Office of General Counsel, completed its presentation of evidence both as to the risks (human, environmental, and economic) from continued Aldrin/Dieldrin usage, the availability of preferable alternative compounds and the projected economic consequences of discontinuation.

It is clear that a great deal of evidence was simply not available to former Administrator Ruckelshaus at the time of his re-evaluation of the suspension issue on December 7, 1972. A brief elaboration of such evidence is set forth below in Section III of this Order. In addition, one cannot ignore events of this summer such as the necessary condemnation of more than eight million Dieldrin contaminated chickens (some of which accumulated levels of Dieldrin as high as 3 ppm in the fat) in the State of Mississippi. This occurrence highlights a major potential problem which will continue to exist as long as these persistent, highly fat soluble compounds continue to be used. While the incident in Mississippi is unique in its staggering proportions, I am informed that it is by no means an isolated incident but affects other industries as well. Whether these incidents are a result of accidents or misuse, or whether they are a direct consequence of the intense agriculture use on feed and food crops, does not of course alleviate the economic consequences which must be borne by the affected industry or the serious potential risks to public health. Indeed, the regular pattern of such occurrences would seem to indicate that as long as Aldrin and Dieldrin continue to be used, such continuing threats to the public safety are inevitable.

III *Evidence in support of suspension*. In remanding the suspension issue to EPA in May of 1972 the Court of Appeals, as previously noted, put special emphasis on the issue of carcinogenicity, asking EPA to elaborate on the nature and extent of such evidence. "Environmental Defense Fund, Inc. v. Environmental Protection Agency," supra, at 538. Consequently a discussion of the limited evidence available at that time, i.e. evidence of liver tumors caused by Dieldrin in a single strain of mouse, constituted the principle rationale supporting the Administrator's finding of a "risk" amounting to a "substantial question of safety" but not "a red light requiring immediate elimination of all dieldrin residues in the diet."[*] (December 7, 1972, Order, at 10, fn. 5). The Administrator did not elaborate further on the risks of other toxic effects nor the issue of benefits or lack thereof.

I am not required here to make an extensive elaboration with findings and conclusions on the multiple issues involved in the cancellation proceeding. As the Court of Appeals observed in "Environmental Defense Fund v. Environmental Protection Agency," supra, at 537, "the function of the suspension decision is to make a preliminary assessment of evidence, and probabilities, not an ultimate resolution of difficult issues." Thus, I will outline with specificity why the best scientific and medical evidence compels suspension at this time.

Specifically, we have learned the following pertinent information:

1. Since the 1970 usage of Aldrin, the last year for which complete use figures were available prior to the issuance of the December 1972 Suspension Order, the use of Aldrin has actually increased from 8.9 million to 11.8 million pounds in 1972. Thus, the continued decline in use that was anticipated at that time has not been realized.

2. For the most recent reporting period of Fiscal Year 1973, the Food and Drug Administration, in its market basket survey, reports that measurable amounts of Dieldrin were found in composite samples of 83 percent of all dairy products, 28 percent of all garden fruits (except tomatoes, green peppers, cucumbers), 96 percent of all meat, fish, and poultry samples and in percentages which range from 12 percent to 42 percent in other food composites of grain and cereal products, potatoes, leafy vegetables, oils, fats and shortening, and fruit. In the normal diet the majority of total Dieldrin intake is due to the residues in dairy products, meat, fish, and poultry. While actual Dieldrin intake levels have shown a slight decline in the market basket survey for the years 1971 and 1972, the percentage of major food category composites found to contain Dieldrin have

---

[*] It should be noted prior to discussion of the evidence that while Aldrin use accounts for nearly 95 percent of the total use of the two compounds, Aldrin is known to break down quite rapidly into its metabolite Dieldrin. Consequently, residues found in the environment are principally Dieldrin residues; and thus the hazards of Dieldrin are generally focused upon. Occasionally the two are used interchangeably.

actually shown a steady increase during this same period.

In addition, air monitoring conducted by this Agency during the years 1970–72 reveals that Dieldrin was detected in over 85 percent of the 3,345 air samples taken nationally, so that respiration must be considered an additional source of human Dieldrin intake.

3. It appears from recent data that virtually every individual in this country has Dieldrin stored in the body. Based on the annual national human monitoring survey conducted by this Agency, tissue samples taken during therapeutic surgery or at autopsy revealed that in 1970, 96.5 percent of all individuals tested had detectable residues of Dieldrin in their adipose tissue, with an average of 0.27 ppm. For the year 1971, 99.5 percent of all those sampled had detectable amounts that averaged 0.29 ppm.

4. Evidence now available indicates that Dieldrin definitely causes significant increases of tumors in two and probably three different strains of mice tested. Moreover, there is positive evidence of increased tumor incidence when Dieldren was fed in low doeses to two different strains of rats as well. Many of these tumors have been diagnosed unequivocally by eminent pathologists as malignant. There is further positive evidence af malignancy based on metastasis to other organs and transplantability into untreated host animals. Dieldrin-caused tumors in both mice and rats appear at a variety of sites within the body, including the liver, lungs, lymphoid tissue, thyroid, uterus and mammary glands. These tumors have resulted at highly statistically significant levels from dietary dosages as low as 0.1 ppm in the diet, which is the lowest dosage ever tested in any animal species. In short, even the lowest levels of Dieldrin produced significant cancerous effects. Furthermore, the evidence indicates that exposure to Dieldrin for periods as brief as several weeks is sufficient to cause highly significant carcinogenic effects in test animals.

This evidence is considerably more extensive than that involving the single strain of mouse discussed in the December 7, 1972, Order by the Administrator. This is not to say that a compound should not be considered carcinogenic because the first and only evidence of carcinogenicity is based on the results of a single experiment in a single strain of one particular test species. Indeed, such evidence generally raises a substantial question of safety requiring commencement of cancellation proceedings. Recent observations made by scientists in the World Health Organization's International Agency for Cancer Research demonstrate that it is unlikely that a compound shown to be carcinogenic in one species will not similarly be carcinogenic when adequately tested in another test species. The more extensive data which have now been developed on the carcinogenicity of Dieldrin confirm and augment the original data from the single strain of mouse. World cancer experts who have testified at the cancellation hearings earlier this year have confirmed the very serious nature of this evidence.

5. While there is no known way of extrapolating absolute conclusions from animals to man, we do know that the basic overall similarity of the experimental animal to man from the standpoint of carcinogenicity is clear in principle. The principle is accepted by U.S. Government Agencies and private health organizations. While recognizing the fact that exposure to even the smallest amount of a carcinogen is no guarantee of absolute safety, scientists at the National Cancer Institute have devised one method for estimating the degree of cancer risk to a particular carcinogen. These estimates are derived from the animal cancer test results. Based upon these calculations and the necessary assumptions, the present estimated average human daily dietary intake of Dieldrin subjects the human population to an extremely high cancer risk.

6. While most of the data with respect to the estimated daily intake of Aldrin/Dieldrin are computed on an average basis, it is obvious that based on differences in dietary composition some segments of the population will greatly exceed that average. In fact, we have now learned from a national dietary survey that young children, particularly infants from birth to one year of age, because of their high dairy product diets, consume considerably more Dieldrin on a body-weight basis than any other age segment of our population. Evidence from laboratory experiments with test animals has shown that the newborn is generally more sensitive to carcinogens. Therefore, infants exposed to Dieldrin may be subjected to a considerably increased risk. It has been shown that in humans Dieldrin is transferred to the fetus during pregnancy. Thus exposure to Dieldrin begins at the earliest stages of life.

7. Evidence based upon human subjects is virtually impossible to obtain. The general human population is continually exposed to a multiplicity of chemicals. A significant "control group" is thus impossible to establish. Moreover, to await the twenty to thirty years of exposure necessary to determine the ultimate effect is only to wait until the damage to an entire generation of humans is complete. We reject the "body count" approach to protection against cancer or other such long term threats to public health. Prediction based on laboratory testing is thus necessary and unavoidable if public health is to be protected.

8. There are additional serious questions as to other toxicological effects demonstrated by these compounds which have a bearing on further human and environmental risks. These include, birth defects caused by Aldrin and Dieldrin in hamsters and mice, adverse effects on learning capabilities in monkeys fed low levels of Dieldrin, adverse reproductive effects caused by Dieldrin in male and female dogs and mice and evidence showing the danger posed to endangered species such as the bald eagle.

9. Finally, there is no agricultural necessity for the major use of these compounds. It is estimated that more than 90 percent of the total usage of Aldrin and Dieldrin is on corn. According to the most recently published U.S. Department of Agriculture statistics, less than 10 percent of the total corn grain producing acreage in the United States is treated with these compounds. On the acreage where Aldrin is used, there are environmentally preferable substitute pesticides, alternative means of pest control or promising substitutes awaiting Federal registration.

The number of additional uses which are actually being defended in the hearing is quite small. For most of these minor uses there also are alternative pesticides which can be utilized. In a few specific instances of very minor uses, there may be no registered alternatives at this time. However, the provision of this suspension order permitting continued use of already formulated Aldrin and Dieldrin products will give some time for the registration of promising environmentally tolerable alternatives, where registrations do not already exist.

As was stated by a subgroup within the U.S. Department of Agriculture reviewing Aldrin/Dieldrin residues in food and feed as far back as December, 1968:

It is pertinent to note an experience of about ten years ago when it was clearly determined by residue studies that aldrin, dieldrin, and heptachlor could no longer be permitted to control grasshoppers on western rangeland because of meat residue problems. The search for nonpersistent alternative insecticides was stimulated and an effective organophosphorous insecticide was found. Thus, a serious food safety problem was eliminated. Agriculture in general would not suffer if aldrin-dieldrin were eliminated from use on agricultural crops.

Having reviewed the above stated pertinent factual data as well as all other available pertinent data, I am persuaded that there exists an "imminent hazard" within the meaning of the statute (as defined by section 2(e) of the FIFRA). It should be noted that during late 1973 and early 1974 the Agency staff presented its evidence on the carcinogenicity of Dieldrin. During this time the manufacturer, through counsel, had its full resources available for extensive cross examination of witnesses. The manufacturer has completed the presentation of most of its evidence on other aspects of the case. While earlier this year it was anticipated that the responsive evidence on carcinogenicity would have already been completed, it now appears that this evidence will be presented during September and October of this year. The cancer experts with whom we have consulted advise us that the rebuttal evidence thus far proffered by Shell is unlikely to be persuasive. Further assessment of the substantiality of this evidence can be made at the expedited hearing, if the registrants request such a hearing.

IV *Effect of order and considerations given thereto.* I find that in light of the evidence above and because of the time this hearing will take in the future, a situation exists in which the manufacture of Aldrin and Dieldrin during the coming months will be "likely to result in unreasonable adverse effects" on man and the environment.[3] In consultation with the sole manufacturer of Aldrin/Dieldrin, the Shell Chemical Company, and its formulators, a determination shall be made as to the precise extent of formulated products currently on hand as of the date of this order. Any stocks of technical grade Aldrin and Dieldrin which have not already been formulated into products may not henceforth be formulated for use in any product other than those uses exempted in the June 26, 1972 order as confirmed in the December 7, 1972 order.

This Agency is not unaware that certain particular uses of pesticides can result in a greater likelihood of unreasonable adverse effects on the environment than others. Such a distinction, however, is particularly difficult to make with respect to the compounds Aldrin/Dieldrin which are so highly persistent, mobile, lipid soluble and capable of exerting such a broad range of toxic effects. Therefore, this order effects all those registered uses for which appeals were duly filed from the June 26, 1972 order (see footnote 1 above).

Finally, I have invoked the new "Special Rule" provision of section 15(b)(2) permitting continued use of those existing stocks of formulated, federally registered products containing Aldrin or Dieldrin. It is held by many of those who have investigated the potential risks and problems attendant to the disposal of consolidated stocks of some toxic materials, such as these pesticides, that it may well be safer environmentally to dispose of them through normal use patterns

---

[3] As further defined by the statute, section 2(bb), the term "unreasonable adverse effects on the environment" can include "any unreasonable risk to man or the environment, taking into account the economic, social, and environmental costs and benefits of the use of any pesticide."

Reproduced from
best available copy.

than to attempt to retrieve the product from the retailer or user, and then to transport, consolidate and either bury or burn remaining supplies. Absent invoking the "Special Rule", this latter alternative is what would be required at present with existing formulated stocks. Additionally, it is my understanding that corn farmers have already applied Aldrin this past spring, so that there remains only limited usage on minor crops during the remainder of the current growing seasons. Permitting use of existing stocks in these situations will not penalize farmers who have already purchased the compounds with the expectation of using them during the remainder of the growing season.

Accordingly, I intend to order the suspension of the registrations and prohibit the production for use of all pesticide products containing Aldrin or Dieldrin which were subject to and for which appeals were duly filed from the Aldrin/Dieldrin cancellation order issued by the Administrator of the Environmental Protection Agency on June 26, 1972 (see footnote 1, above). In the absence of a request for an expedited hearing, this order shall be effective 5 days after receipt by affected registrants.

Dated: August 2, 1974.

RUSSELL E. TRAIN,
Administrator.

[FIFRA Dockets No. 145, etc.]

SHELL CHEMICAL COMPANY, ET AL.

PRELIMINARY STATEMENT REGARDING RECOMMENDED DECISION

These are consolidated proceedings under the Federal Insecticide, Fungicide, and Rodenticide Act, as amended (7 U.S.C. 136 et seq., 1973 Supp.). Pursuant to section 6(c) of the act (7 U.S.C. 136d(c)), the Administrator, on August 2, 1974, issued a notice of intention "to suspend the registrations and prohibit the production for use of all pesticide products containing Aldrin or Dieldrin which are subject to and for which appeals were duly filed from the Aldrin/Dieldrin cancellation order issued by the Administrator of the Environmental Protection Agency on June 26, 1972."[1] The notice of suspension also contained detailed findings pertaining to the question of "imminent hazard" as required by the act.[2]

"Unreasonable adverse effects on the environment" is defined in the act to mean "any unreasonable risk to man or the environment, taking into account the economic, social, and environmental costs and benefits of any use of any pesticide." (7 U.S.C. 136(bb)).

Shell Chemical Company, the sole manufacturer of the pesticides involved, filed timely objections to the notice of intention to suspend and subsequently 22 other registrants also filed objections thereto.[3] In addition, the Secretary of Agriculture of the United States, Environmental Defense Fund, Inc., the National Audubon Society, and Florida Citrus Mutual were granted leave to intervene herein pursuant to § 164.121(e) of the rules of practice (38 FR 19371, 19378).

Section 6(c)(1) of the statute further provides that "No order of suspension may be issued unless the Administrator has issued or at the same time issues notice of his intention to cancel the registration or change the classification of the pesticide." By PR Notice 71-4, dated March 18, 1971, and issued by the Acting Director of the then Pesticides Regulation Division, after prior piecemeal cancellations of registrations of pesticides containing the insecticides aldrin and dieldrin, the registrations under the act of all registrations of products containing aldrin and dieldrin were cancelled. Of the 86 registrants who, in effect, appealed the cancellation of their registrations by PR Notice 71-4, 2 requested a public hearing and 84 registrants requested that the matter be referred to an advisory committee selected by the National Academy of Sciences, which they could then do under the statute. The cancellations involved were not effective pending the outcome of such appeals. The Aldrin/Dieldrin Advisory Committee to the Administrator issued a report March 28, 1972, recommending, in part, that certain uses of the pesticides involved be disallowed, that enumerated uses thereof are "valuable and not harmful," that further studies be conducted in specified areas and that a further review be conducted in the future.

By a Determination and Order dated June 26, 1972, then required by the statute, the Administrator affirmed the cancellation of the registrations of all products containing aldrin or dieldrin except with respect to those registered uses involving the dipping of roots or tops of nonfood plants, subsurface ground insertions for termite control and mothproofing by manufacturing processes which utilize the pesticide in a closed system, which uses the Administrator found to "pose *de minimus* risks." The Administrator therein deferred decision on the suspension, as distinguished from the cancellation, of the aldrin and dieldrin registrations.

Section 4c of the act (7 U.S.C. 135b(c)) then provided that administrative appeals from the decision of the Administrator to maintain cancellations in effect may be taken within 60 days from the date of such decision. Appeals therefrom were taken by the filing of objections thereto and request for a public hearing by 38 registrants.

The Administrator, by a Determination and Order dated December 7, 1972, in part, consolidated into the cancellation proceedings petitions dealing with tolerances of aldrin and dieldrin pursuant, in effect, to sections 406 and 408 of the Federal Food, Drug, and Cosmetic Act (21 U.S.C. 346 and 346a). The Administrator also therein declined to suspend the registrations involved, clarified his prior order with respect to permitted uses and, in effect, lifted the cancellations of registrations for manufacturing use only. Oral hearing in the cancellation proceedings commenced August 7, 1973, and was in progress when the notice of intention to suspend was issued.[4]

On August 7, 1974, Herbert L. Perlman, Chief Administrative Law Judge, Environmental Protection Agency, was appointed the Presiding Officer in the suspension proceedings. Prehearing conferences were held August 7, 8, 9 and 13, 1974, and the hearing herein commenced August 14, 1974. The registrants filing objections to the notice of intention to suspend subsequent to the filing of objections by the Shell Chemical Company were consolidated into the proceeding instituted by Shell pursuant to § 164.121(f) of the rules of practice and evidence received in the cancellation proceedings was incorporated by reference into the suspension proceedings by the agreement of the parties. In addition, respondent did not present evidence herein with respect to the matters contained in paragraph 8 of the August 2, 1974 notice of intention to suspend dealing with toxicological effects of aldrin and dieldrin other than cancer, and danger posed to endangered species.

The Administrator ordered that the hearing herein take no longer than 15 hearing days and the hearing closed September 12 1974. The active participants at the hearing were represented by the following:

William D. Rogers, Andrew S. Krulwich, David H. Lloyd and Linda Blumenfeld, Attorneys at Law, Washington, D.C., representing Shell Chemical Company,

Raymond W. Fullerton and Richard S. Wasserstrom, Office of the General Counsel, United States Department of Agriculture, representing Intervenor Secretary of Agriculture of the United States; and John A. Knebel, General Counsel, United States Department of Agriculture, who presented one witness and made oral argument for this intervenor,

William A. Butler and Jacqueline M. Warren, Attorneys at Law, Washington, D.C., representing Intervenors Environmental Defense Fund, Inc. and the National Audubon Society, and

John C. Kolojeski, William E. Reukauf, Timothy L. Harker, Edward Lyle, and John W. Lyon, Office of the General Counsel, Environmental Protection Agency, representing respondent Assistant Administrator, Environmental Protection Agency.

Subsequent to the close of hearing the parties filed briefs and I hereby submit my recommended decision within the exceedingly short period of time provided by the rules of practice.

FINDINGS OF FACT

1. The registrants in these consolidated suspension proceedings are as follows:

Agway Inc., a corporation whose address is Box 1333, Syracuse, New York;
AMOCO Oil Company, a corporation whose address is 200 East Randolph Drive, Chicago, Illinois;
Arlenge Laboratories, Inc., a corporation whose address is 175 Pearl Street, Brooklyn, New York;
Borden, Inc., a corporation whose address is 60 West Broad Street, P.O. Box 2478, Columbus, Ohio;

---

[1] As explained in the August 2, 1974 notice, a final order of suspension in these consolidated proceedings would not include the 3 uses permitted by the June 26, 1972 order, that is, restricted termite use, the dipping of roots and tops of nonfood plants and use in a totally effluent-free mothproofing system. Also, the August 2, 1974 notice of suspension permitted, pursuant to section 15(b)(2) of the act (7 U.S.C. 136m(b)(2)) the "use or sale of existing formulated stocks of pesticides containing Aldrin or Dieldrin which were on hand as of the effective date of the suspension order."

[2] Section 6(c)(1) of the act provides that "If the Administrator determines that action is necessary to prevent an imminent hazard during the time required for cancellation or change in classification proceedings, he may, by order, suspend the registration of the pesticide immediately." The term "imminent hazard" is defined to mean, in part, "a situation which exists when the continued use of a pesticide during the time required for cancellation proceeding would be likely to result in unreasonable adverse effects on the environment * * *" (7 U.S.C. 136(1)).

[3] It is not clear that all of the additional registrants filed timely objections and are properly parties to these proceedings. However, respondent has failed to file motions to dismiss in this regard and we are not in possession of the facts to enable us to decide this issue.

[4] By August 2, 1974, over 24,000 pages of transcript and many thousand of pages of exhibits, including the witnesses' direct testimony, were adduced in the consolidated cancellation proceedings.

Borderland Products, Inc., a corporation whose address is 560 Fulton Street, P.O. Box 366, Buffalo, New York;

Bonide Chemical Company, Inc., a corporation whose address is Utica, New York;

C. J. Martin Company, a company whose address is 606 West Main Street, P.O. Box 1089, Nacogdoches, Texas;

Chevron Chemical Company, a company whose address is 200 Bush Street, San Francisco, California;

Coastal Chemical Corporation, a corporation whose address is Evans Street, Extension, P.O. Box 856, Greenville, North Carolina;

Colorado International Corp., a corporation whose address is 5321 Dahlia Street, Commerce City, Colorado;

Dexol Industries, a company whose address is 1450 West 228th Street, Torrance, California;

Farmland Industries, Inc., a corporation whose address is P.O. Box 7305, Kansas City, Missouri;

FCX Inc., a corporation whose address is P.O. Box 2419, Raleigh, North Carolina.

Helena Chemical Company (Midsouth Division), a company whose address is P.O. Box "N", West Helena, Arkansas;

Key Laboratories, Inc., a corporation whose address is Baskins Crossing, Largo, Florida;

McLaughlin Gormley King Company, a company whose address is 1715 S.E. Fifth Street, Minneapolis, Minnesota;

Riverside Chemical Company, a company whose address is P.O. Box 17119, Memphis, Tennessee;

Shell Chemical Company, a division of Shell Oil Company, a corporation, whose address is 2401 Crow Canyon Road, San Ramon, California;

Southern Agricultural Insecticides, Inc., a corporation whose address is P.O. Box 218, Palmetto, Florida;

Stauffer Chemical Company, a company whose address is 1200 South 47th Street, Richmond, California;

Stephenson Chemical Company, Inc., a corporation whose address is P.O. Box 87188, College Park, Georgia;

Stevens Industries, Inc., a corporation whose address is Dawson, Georgia; and

Triangle Chemical Company, a company whose address is P.O. Box 4528, 206 Lower Elm Street, Macon, Georgia.

2. The intervenors in these consolidated suspension proceedings are the Secretary of Agriculture of the United States, Environmental Defense Fund, Inc., National Audubon Society, and Florida Citrus Mutual. The respondent herein is the Assistant Administrator, Environmental Protection Agency.

3. Aldrin is the common name of a chemical compound approved by the International Organization for Standardization (except in Canada, Denmark and U.S.S.R.) and by the British Standards Institution for a material containing not less than 95 percent of 1,8,9,10,11,11-hexachloro-2,3-7, 6-endo-2,7,8-exo-tetracyclo $[6.2.1.1^{3,6}.0^{2,7}]$ dodec-4,9-diene. In Canada, aldrin refers to the pure compound, known as HHDN in Great Britain. It was introduced in the United States in 1948 by Julius Hyman and Company as Compound 118 under the trademark Octalene. In December 1949, the insecticide was given the common name "aldrin" by the Interdepartmental Committee on Pest Control of the United States Department of Agriculture. It has been used as a broad spectrum insecticide on a variety of crops and in a wide variety of locations and situations. Its insecticidal action was first described by Lidon under patent number 2,635,977 (this was transferred to Shell Development Company in 1953), and Schmerling had patent number 2,911,477 (transferred to Universal Oil Products in 1959). The physical properties of the compound are as follows:

(a) As a pure compound, it is a white crystalline odorless solid, with a molecular weight of 364.93.

(b) It has a melting point of 104–104.5° Centigrade.

(c) Its vapor pressure is 2.31×10^{-6}mm of Mercury at 20° Centigrade.

(d) It is slightly soluble in water (0.0027 mg/100 ml or 2.7×10^{-6} grams per 100 milliliters of water).

(e) It is lipophilic, having a strong attraction for fats, and is fat soluble.

(f) Its colubility in various substances is as follows:

Pentane—3 grams per 100 milliliters at 25°C.

Ethanol—5 grams per 100 milliliters at 25°C.

n-Butanol—9 grams per 100 milliliters at 25°C.

Butanone—24 grams per 100 milliliters at 25°C.

Amylacetate—30 grams per 100 milliliters at 25°C.

Acetone—66 grams per 100 milliliters at 25°C.

Benzene—83 grams per 100 milliliters at 25° C.

Xylene—92 grams per 100 milliliters at 25°C.

Ethylenedichloride—105 grams per 100 milliliters at 25°C.

Carbon Tetrachloride—105 grams per 100 milliliters at 25°C.

(g) It is stable in the presence of organic and inorganic alkalies.

(h) Oxidizing agents and strong acids attack the unchlorinated ring.

(i) Upon prolonged storage, there is a slow formation of Hydrochloric acid (HCl) which causes it to be corrosive.

(j) The technical product is a tan to dark brown solid with a melting range of about 49 to 60°C.

(k) It is a non-systemic and persistent insecticide.

4. Dieldrin, a manufactured product and a metabolic degradation product of aldrin, is the common name approved by the International Organization for Standardization (except in Canada, Denmark and the U.S.S.R.) and by the British Standards Institution for a material containing not less than 85 percent of 1,8,9,10,11,11-hexachloro - 4,5 - exo-epoxy-2,3-7,6-endo-2,1 - 7,8 - exo - tetracyclo $[6.2.1.1^{3,6}0^{2,7}]$ dodec-9-ene. In Canada dieldrin refers to the pure compound, known as HEOD in Great Britain. It is used as a broad spectrum insecticide and was first introduced in 1948 by Julius Hyman and Company as Compound 497 under the trade name of Actalox. In December, the insecticide was assigned the common name "dieldrin" by the Interdepartmental Committee on Pest Control. It is classified as a non-systemic and persistent insecticide of high contact and stomach activity to most insects. U.S. patents were granted to Soloway with the patent registration number of 2,676,131. This was transferred to the Shell Development Company in 1954. Another U.S. patent was issued to a Payne and Smith, patent number 2,776,301 which was transferred to Shell Development Company in 1957. A British patent number 794,373, was assigned to N. V. Bataafsche in 1958. Some of the physical properties are as follows:

(a) The pure compound is a white odorless crystalline solid with a molecular weight of 380.93.

(b) Its melting point is 175–176° Centigrade.

(c) Its vapor pressure is 1.78×10^{-7} millimeters of mercury (Hg) at 20° Centigrade.

(d) It is lipophilic, has a strong attraction to fats, and is fat soluble.

(e) Its solubility in various substances is as follows:

Oil, Standard No. 10—1.3 grams per milliliter at 30° C.

Hexane—2.5 grams per milliliter at 30° C.

Methanol—3.4 grams per milliliter at 30° C.

Acetone—35.4 grams per milliliter at 30° C.

Benzene—36.9 grams per milliliter at 30° C.

(f) It is slightly soluble in water, 0.186 milligrams in 100 milliliters of water to say it in another way, 1.86×10^{-4}g/100 ml.

(g) Dieldrin is more stable than aldrin as indicated by its stability when exposed or combined with alkali and mild acids.

(h) The technical product is buff to light brown flakes with a setting point not below 95° C.

5. Beginning in 1950, Shell Chemical Company became the sole national distributor for aldrin and dieldrin and Julius Hyman and Company remained the sole manufacturer. In May, 1952, Julius Hyman and Company was amalgamated with Shell as the Julius Hyman and Company Division of Shell Chemical Corporation. From 1952 until 1967, Shell sold only technical aldrin and dieldrin to pesticide formulators who in turn made it up into emulsible concentrate, dust, wettable powder or granular formulations for sale under their own company's brand name. Beginning in 1967, Shell started selling formulated product under the Shell brand name. By 1972, only 11 percent of the total aldrin and dieldrin sold was sold as technical product for use in non-Shell branded formulations.

6. Aldrin and dieldrin are toxic to humans. In the instance of aldrin, poisoning may occur by ingestion, inhalation, and/or skin absorption. Severe symptoms may result from ingestion or percutaneous absorption of 1 to 3 grams, especially in the presence of liver disease. Renal damage, tremors, ataxia, convulsions followed by C.N.S. depression, respiratory failure and death can occur from acute exposures. Chronic exposures over a prolonged period may cause at least hepatic or liver damage.

7. (a) Approximately 1.5 million pounds of aldrin were sold in 1950, the year it was introduced, practically all of this for use on cotton. Sales for use on cotton continued to account for a major portion of the total aldrin sales until the mid-1950's when the superior effectiveness of dieldrin against the boll weevil became widely known. Sales for use on cotton, particularly in the southeast, where quick effectiveness between the many rain showers is a necessity, continued until the mid-1960's. In 1954, cotton accounted for 30 percent of the total sales whereas in 1963, the last year of any real cotton use, it was less than 1 percent.

(b) Two ounces of aldrin per acre diluted in diesel oil was an effective and economical grasshopper insecticide and it was adopted for all Federal cooperative grasshopper control programs. By 1954 approximately 4 million acres had been treated with aldrin. Aldrin remained the insecticide of choice until the late 1950's when dieldrin at 0.5 ounce per acre became the insecticide of choice and was used until the mid-1960's. In 1954, use for grasshopper control programs accounted for approximately 15 percent of total sales but decreased to less than 1 percent in the early 1960's. In addition to the use of aldrin in the Federal grasshopper control programs, substantial quantities of aldrin (and dieldrin) were sold for use in other Federally-sponsored programs from 1954 to the late 1960's. These included eradication programs for Japanese beetle, European chafer, white fringed beetle, and Imported Fire Ants. Beginning in the late 1950's, dieldrin became the compound of choice for all of these programs, but some aldrin was continued for the Japanese beetle programs.

(c) Other early uses which accounted for substantial quantities of aldrin, and later were determined to lead to high residue in foods and their by-products, sometimes used as animal feeds, were soil applications on land planted to potatoes, peanuts and sugar beets. In 1954, these uses accounted for approximately 13 percent of the total used. The total pounds used annually remained fairly constant at approximately one-half million pounds through 1962. Soil use on potatoes and peanuts was withdrawn in 1963 but use on sugar beets continued until 1967.

(d) Until 1955, cotton was the principal use crop for aldrin. Corn soil usage took the lead that year and has been the main single use since. As of 1971, the estimates showed that corn soil usage accounted for 80 percent of the total sales for this product. Other end uses and their percent of the total were as follows: Termite and PCO, 14 percent; rice seed treatment, 3 percent; citrus soil use, 1 percent; other small grains, corn and vegetable seed treatments, 1 percent; and miscellaneous soil applications including on tobacco, vegetables, strawberries, 1 percent. Some of the principal end uses of aldrin for 1954, 1964, 1968 and 1971 were as follows:

ALDRIN END USE ESTIMATES—1000 LBS.

	Year			
	1954	1964	1968	1971
Cotton (foliage)	834	19		
Corn (soil)	804	10,191	12,089	9,410
Grasshoppers	476	20		
Potatoes (soil)	289			
Peanuts	81			
Citrus (soil)		35	200	150
Sugar beets		60		
Seed treat (except rice)	6	80	150	130
Rice seed treatment		235	472	288
Japanese beetle		13		
White-fringed beetle	10			

The end use estimate of aldrin under corn is 8.8, 6.9 and 7.6 million pounds during 1972, 1973 and 1974, respectively.

(e) A continued gradual decline in aldrin sales in the future may occur as corn rootworm resistance moves eastward through Indiana and Ohio. Also seed corn maggot resistance to aldrin may also spread outside the Iowa-Illinois area into other corn-producing states.

8. (a) Dieldrin was first used as a spray or dust on cotton for boll weevil control. Because of its effectiveness against all cotton pests except the lepidopterous species, it was widely used in Texas and the Mississippi Delta area. Dieldrin required fewer applications because of its residual effectiveness and was applied every seven to ten days as the infestations warranted. Practically all of the 1951 sales of dieldrin were for use on cotton. This use peaked in 1955 when slightly more than one million pounds were sold for cotton insect control. The boll weevil became resistant to all chlorinated insecticides in the late 1950's, and only minor quantities were sold in the 1960's.

(b) Forage crop uses, particularly for alfalfa weevil control when this insect moved into the northeastern United States, accounted for approximately one million acres being treated annually during the mid to late 1950's and early 1960's. Armyworm, which attack sporadically, accounted for several million acres of small grains being treated in the Midwest in the early 1960's. Dieldrin as well as aldrin was used in the Federal grasshopper control programs until the mid-1960's. Other forage crop pests of lesser importance which were controlled by foliage applications of dieldrin were chinch bugs and grasshoppers attacking corn and small grains and the pale Western cutworm, which attacked small grains in the Rocky Mountain states area.

(c) Dieldrin was also very effective against houseflies and mosquitoes until these pests became resistant. It was also effective against deer flies, sand flies, black flies and many other public health pests which were injurious and annoying to man and animals. During the 1950's and into the 1960's, dieldrin was used both by individuals as well as state and local agencies to control these pests. These uses led to high residues of dieldrin in some aquatic environments.

(d) Late in the 1950's, it was found that dieldrin was a very effective material to permanently mothproof woolen goods, particularly carpets. If used in the hot acid dye bath, dieldrin would be taken into the wool fiber and "locked" into the fiber. After registration was granted, many of the woolen mills in the United States started using dieldrin. Approximately 250 thousand pounds of dieldrin were used annually until Shell Chemical Company withdrew the registration in 1970 when it was determined that some dieldrin would remain in the dye bath effluent which was discharged into streams and rivers.

(e) As with aldrin, government-sponsored eradication programs for Japanese beetle, white-fringed beetle, European chafer, Imported fire ants and alfalfa snout beetle took considerable quantities of dieldrin from the mid-1950's through the late 1960's. Probably the biggest program was for white-fringed beetle where usage has averaged more than 100 thousand pounds annually since 1955.

(f) The overall use of dieldrin has dropped from a peak of 3.6 million pounds in 1956 to approximately 600 thousand pounds today. As of 1971, the end use sales estimates showed the following percent of the total sales for the following uses: Termites and PCO, 44 percent; fruit (foliage), 20 percent; seed treatment, 14 percent; vegetables, 13 percent; and miscellaneous uses including on tobacco, sweet potatoes, etc., 9 percent. Sales volumes for 1954, 1964, 1968 and 1971 for some of the principal end uses at that point in time were as follows:

DIELDRIN END USE ESTIMATES—1,000 LBS

	Year			
	1954	1964	1968	1971
Cotton (foliage)	757	20	1	
Public health	62			
Government programs	133	205	104	
Fruit (foliage) (plum curculio)	202	405	217	120
Mothproofing		320	158	
Small grains (foliage)	175	150		
Small Package (home and garden use)		227	34	2

9. The domestic sales of aldrin and dieldrin from 1950 through July 1, 1974, including consumer/specialty sales but excluding sales to the World Health Organization and the Agency for International Development are as follows:

Year	Aldrin (1,000 lbs)	Dieldrin (1,000 lbs)
1950	1,456	0
1951	2,268	185
1952	814	750
1953	1,234	1,135
1954	2,903	1,777
1955	4,372	2,585
1956	6,495	3,635
1957	2,431	2,673
1958	4,971	3,074
1959	5,366	3,005
1960	8,109	2,650
1961	9,958	2,764
1962	10,896	2,950
1963	12,152	2,655
1964	12,093	2,052
1965	14,278	1,514
1966	19,327	1,908
1967	13,092	1,473
1968	12,920	1,222
1969	9,902	1,206
1970	8,509	749
1971	11,615	705
1972	11,568	740
1973 (to July 1)	[illegible],721	432
1973 estimated (to Dec. 31)	(1[illegible],000)	(576)
1973	9,900	
1974 (to July 1)	8,700	

10. The Aldrin/Dieldrin Advisory Committee appointed by the Administrator issued a report March 28, 1972, which contained the following conclusions and recommendations:

Conclusions. We find evidence of human injury from present or past use of aldrin or dieldrin. Nevertheless the facts that fairly low levels of dieldrin can cause cancer in mice and interfere with reproduction in some birds are matters for concern, and point to the need for more careful evaluation of the hazard to man. There is clear evidence that past usages have been deleterious to wildlife. Several such past usages have been voluntarily abandoned by Shell Co. Nevertheless, we feel that we must strive to find alternate methods of pest control, including nonchemical methods, for all compounds which lead to persistent residues in humans or wildlife, even when such residues are not demonstrably harmful. How can we move towards this objective. When aldrin or dieldrin can be safely and economically replaced by nonpersistent pesticides they should be so replaced. Several practices which can readily lead to damaging effects upon non-target organisms should be abandoned now in spite of the difficulty of economic replacement, including all applications which lead to contamination of aqueous environments such as rice fields and waterways.

The direct application of aldrin or dieldrin to soils leads to negligible leaching or other transfer from these soils, and environmental contamination is thus very small except where substantial erosion takes place. One of the few studies to estimate the amount which volatilized indicates that 3 percent escapes this way, and thus contaminates the environment directly (we would like to see more extensive data upon this point).

Recommendations. The following recommendations are designed to build a basis of facts on which permanent recommendations can be formulated, and to eliminate now those uses of aldrin or dieldrin which result in significant environmental contamination (especially to waterways). We believe that applications directly to soil or to materials buried in soil (e.g. termite control in foundations, and seed treatments when properly applied) lead to little subsequent movement of these insecticides, and should be permitted.

In the following recommendations, we use the term "experts" and "acknowledged authorities" advisedly. The EPA must seek contractual or other arrangements with individuals and institutions accepted as authorities by their peers in the country at large.

1. A committee of experts in chemical carcinogenesis should be formed to propose specific experiments and to agree upon suitable protocols to provide a firm indication of the extent of carcinogenic hazard. These experiments should include studies (in at least two vertebrate species) on the effects on the progeny of mothers fed dieldrin during pregnancy and nursing, the progeny also being fed dieldrin thereafter.

2. The economic consequences of total withdrawal of aldrin and dieldrin should be explored in depth: On all major crops, actual experimental studies must be performed to obtain new, reliable data provided by acknowledged authorities, and should include studies with and without alternative nonpersistent pesticides, over a series of years, and in appropriately distributed geographical areas.

3. The fraction of aldrin and dieldrin which escapes by volatilization following application to a variety of soils, under conditions of application and treatment levels commonly used in pest control, should be measured by acknowledged authorities.

4. Monitoring stations should be established in the U.S. and abroad, at which air and water samples can be taken at fixed places over a series of years, and analyzed by unambiguous procedures for aldrin and dieldrin. The intent is to study whether the restrictions we propose do indeed lead to a progressive removal of these compounds from the environment. Agreement should also be sought amongst a group of experts for unambiguous procedures for determination of aldrin and dieldrin in extracts of air, soil, water, food and human and nonhuman tissues. Such procedures should be standardized in the U.S. and preferably internationally as well.

5. The following uses of aldrin or dieldrin should be disallowed.

(a) All applications by aircraft.

(b) All foliar spraying or dusting.

(c) Moth proofing by the hot acid dye bath method or related methods in which residues are discharged into waterways or settling ponds.

(d) All uses, whether by homeowners or pest-control operators, in homes, barns, poultry operations or other structures occupied by humans or livestock.

(e) Use upon turf (including lawns and non-grazing grassed areas) except as supervised or controlled by trained or licensed pest-control operators, greenskeepers and nurserymen.

(f) Any use which involves application to streams, ponds, lakes, flooded areas or any other aquatic environments.

6. Specific uses of aldrin and dieldrin which we believe to be valuable and not harmful include:

(a) Direct applications to soils.

(b) Seed treatments, when the treated seed is labelled "not for food use".

(c) Dipping of plant roots or tops during transplantation.

(d) Treatment of foundations, by current procedures, for termite control.

(e) Use of treated hot-caps.

7. Because our recommendations are based upon evidence which, although the best available, is still not complete; we recommend that the environmental and economic effects of the proposed restrictions be reviewed 5 years after their imposition. By that time, the completed results of recommendations 1, 2, 3 and 4 should be available.

11. Cancer is a major and increasing cause of death and morbidity in man. It imposes upon society an immense burden of death, suffering, and economic loss.

12. Chemical carcinogensis has two key characteristics, irreversibility of effect, and long latent period between initial exposure and manifestation of symptoms. In principle, no dose of a chemical carcinogen is too small to induce cancer in susceptible individuals. Some cancers do not develop until late in life—in man, usually 20 and sometimes 30 or 40 years after initial exposure.

13. Chemicals known to cause cancer in man have been identified only through epidemiological studies, either in the general public, or in occupationally exposed workers. In the case of aldrin/dieldrin, epidemiological studies in the general population are not possible because there are no clearcut differentials of exposure and because the period of exposure has been too short. A study of occupationally exposed workers, carried out by the Shell group of companies, is of no value, from an epidemiological standpoint, as a carcinogenicity study because the number of workers studied was too small, the period of observation was too short and only active male workers were studied. As with most chemicals, it is therefore necessary to rely on experiments with animals to determine the potential carcinogenic hazard of aldrin/dieldrin to man.

14. The use of experiments with animals to screen chemicals for potential carcinogenic hazard to man is accepted by the scientific community and by public policy-making agencies in the United States. Chemical carcinogenesis in animals provides a very close parallel to chemical carcinogenesis in man. All chemicals known to cause cancer in man except arsenic which is under study also cause cancer in animals, especially rats and mice. The pathological development of chemically induced tumors in animals and in man is very similar. However, human populations are more variable than the strains of animals usually used in laboratory tests, and some individuals are likely to be correspondingly more susceptible.

15. Chemical carcinogenesis is a specific biological process which is induced by only a relatively few classes of chemicals. It is not true that all chemicals induce cancer at sufficiently high doses. Most, probably all, chemical carcinogens that have been adequately tested cause cancer in more than one species of animal. It is not true that there are "species-specific" carcinogens. Also, it is not true that there are "strain-specific" carcinogens, but some strains of mice are especially susceptible to induction of certain kinds of tumor.

16. Transplantability of tumors and/or metastasizing to other organs provide proof that chemically induced tumors are "malignant"; however, all chemical tumorigens should be regarded as potential carcinogens.

17. Guidelines for conducting acceptable experiments on chemical carcinogenesis in animals have been recommended by expert professional committees. The mouse and the rat are the preferred experimental animal species, both because their relatively short lifespan permits lifetime testing within a reasonable period of time, and because the pathological development of tumors in these species is particularly well known and understood.

18. A number of adequately conducted experiments have shown conclusively that aldrin and/or dieldrin induced cancer in 5 different strains of mice, and, perhaps, in the rat.

19. Reported carcinogenicity tests with aldrin and dieldrin in dogs and monkeys were carried out for too short a period to draw any definite conclusions, but precancerous lesions were observed in the livers of the dogs. No adequately conducted carcinogenicity test with aldrin or dieldrin in any species of animal has given negative results.

20. In the experiments with mice, aldrin and dieldrin induced cancer primarily in the liver, but in some experiments significant incidence of cancers of the lung and other organs was reported.

21. Tumors produced by aldrin and dieldrin in mice have been diagnosed by expert pathologists as unequivocally malignant. In some experiments tumors metastasized to other organs, or were successfully transplanted to other hosts, providing further proof of malignancy. In at least some experiments, malignant tumors produced by aldrin and dieldrin significantly shortened the lifespan of the experimental animals. In the most extensive series of experiments, carried out by Shell research scientists, the incidence of liver and other tumors in mice was clearly dose-related. A significant increase in the incidence of liver and other tumors was observed at the lowest dose tested, 0.1 ppm in the diet.

22. Even a limited exposure to aldrin/dieldrin for only a few weeks early in life led to a significant increase in liver tumors in mice, despite cessation of exposure.

23. None of the reported experiments involved exposure of the experimental animals to aldrin/dieldrin prior to weaning, although younger animals and fetuses *in utero* are likely to be more susceptible to these agents.

24. Dieldrin induces enzymes in the liver which may activate certain environmental carcinogens. A threshold level of dietary dieldrin for induction of these enzymes in man is not known.

25. There is no scientific basis for the existence of a "threshold" or "no-effect" level of exposure of an animal population to a chemical carcinogen. It is impossible to establish a "safe" level of exposure of aldrin/dieldrin to man.

26. Aldrin/Dieldrin have been found to be carcinogens in the mouse as a result of adequately conducted tests in laboratory conditions. They pose a carcinogenic hazard to man.

27. Many kinds of insects spend at least part of their lives in the soil. Of the thousands of insects in or on our soils, only 20 or so are classed as pests of corn. Except for a few species, they are general throughout the corn-growing areas of the United States. While most of the impotrant soil insect pests are found over broad areas, usually only one or a few at a time are of significant economic importance in an individual field. The area, population dynamics, weather, soil type, crop rotation and general agronomic practices will influence the buildup of individual destructive species.

28. A common characteristic of all soil insects is their four-stage life cycle: (1) Egg, (2) larvae (worm or group), (3) pupae (resting stage) and (4) adult (beetle, moth or fly). Eggs are laid by the adult female in areas suitable to that species. Northern corn rootworm female beetles will lay their eggs in cornfields. Female "click" beetles (adult wireworms) usually seek out grassy areas so the young larvae will have sufficient food. However, in Iowa an annual species has been reported to lay eggs in only the bare spots in fields. Where eggs are laid plays an important role in what insects will be present in the spring corn crop as farmers can plant corn following many crops or sods. Eggs hatch into larvae which are commonly called grubs, worms or maggots. With the group called soil insects, this is the stage that usually causes the most damage except for most notably the seed-corn beetle. Most of the larvae, with the exception of the Northern

and Western corn rootworms, have food preferences other than corn. Most are general feeders and when their main food supply is removed they readily adapt to corn. As larvae mature they enter the pupal stage of growth. It's here they complete the change from larvae to adult beetles, or moths or flies. A few of the adult soil insects are also destructive.

29. The corn soil insects which presently can or do cause injury of economic significance are as follows:

(a) *Wireworms: Melanotus sp., Conoderus sp. and Horistonotus sp. and other species of the family Elateridae.* Wireworms species attacking corn may differ some in specific areas but in general they all cause similar damage to seed and young plants. *Melanotus sp.* are most common throughout corn areas and pose the most problems for corn growers. Most of the damaging wireworm species have a life cycle from egg to adult of 2–6 years. The life span appears to be longer (4–6 years) in colder climates and shorter (2–3 years) in southern areas. The *Conoderus sp.* is an annual wireworm laying eggs in grain stubble which has not been second cropped. These wireworms are most prevalent in the southeastern United States but are becoming more of a problem in the central Corn Belt. Adult wireworms (click beetles) show a preference for sod areas and eggs may be laid in pastures, grain stubble, hay fields, weedy row crops and other grassy areas. When sod or other grassy areas are tilled for corn the next spring, the worms feed on the corn seed and young corn plants as their other food diminishes with the elimination of weeds and grass. Because eggs are laid each year in grassy fields, wireworms with more than a one-year cycle may be present in any stage. When populations are heavy they may completely destroy not only the original planting but subsequent replantings. Wireworms like and need moist soil and will tend to follow the moisture table in the ground. In a wet spring they will be more of a problem than in a dry one. Wireworms will tunnel into newly planted seed and kill the germination. They will also bore into the base of young corn plants below ground killing the growing point in the corn plant. The newly-emerged plant starts to wilt and die from the center out and finally the entire plant dies or produces suckers which bear no ears. In large numbers, entire fields can be lost. Planter box treatments and row treatments of aldrin are not as effective as broadcast applications and may not provide adequate control under population stress.

(b) *Cutworms.* Black cutworm, *Agrotis ypsilon* (Rottenburg); Glassy cutworm, *Cyrmodes devastator* (Brace); Bronzed cutworm, *Nephilodes emmedonius* (Crawer); Dingy cutworm, *Feltia subgothica* (Haworth); Bristley cutworm, *Lacinyolia renigera* (Stephens); Clay-backed cutworm *Agrotis gladiaria* (Morrison); Sandhill cutworm, *Euroa deteroa* (Walker). The black cutworm is by far the most widely found and the most damaging. Most of the problem species are surface feeders except for the glassy cutworm which is a true subterranean cutworm. Cutworms will generally feed on the newly-sprouted plants. Moisture in the soil and atmosphere conditions help to control the feeding pattern. When the soil is moist or wet and nights are cool with high humidity, the cutworms will feed on the surface cutting off the corn plants. As the soil dries the cutworms may not surface, feeding only below ground, living in the moist soil. Much of the life cycle and biological history of the cutworms is still unknown. However, in general, they tend to overwinter as nearly full-grown larvae. Adult moths tend to lay eggs in grassy, wet areas. Black cutworms not only overwinter as larvae but migrate into the Corn Belt area from the south in March and April. Cutworm damage is generally associated with poorly drained river bottom land, heavy soils and low wet spots in upland fields. It is also more extensively found in first year corn following sod or legumes. Failure to notice a cutworm problem early may result in a lost field or part of a field that must be replanted.

(c) *White grubs: Phyllophaga or Hachnostenna spp.* These are the most common grub pests. They are the larval form of the common May and June beetles. The beetles prefer grassy areas such as pastures, soil bank land and hay fields. These differ from annual grubs by having life cycles that take 2–4 years to complete. Three-year cycles are most common. White grubs appear most often in cornfields when sod ground or grassy areas are spring plowed. With their 2–4 year life cycle, they can pose a problem to the farmer more than one year. However, the most destructive damage occurs the first year after sod. Damage comes in the form of plants wilting and "drying up." The larvae prune the roots and the plant literally dies of thirst.

(d) *Corn Rootworms.* Northern Corn Rootworm, *Diabrotica longicornis* (Say); Western Corn Rootworm, *Diabrotica virgifera* (Le Conte); Southern Corn Rootworm, *Diabrotica undecimpunctata howardi* (Barber). The Northern corn rootworm inhabits the entire Corn Belt while the Western can be found in damaging numbers in Colorado, Nebraska, Kansas, South Dakota, Minnesota, Iowa, Missouri, Illinois, Indiana, and Wisconsin. The first Western beetles were found in Indiana in 1971. Southern corn rootworms migrate north each year and are usually more of a problem in the southern area of the Corn Belt or in southern corn-producing areas. Northern and Western corn rootworm adults lay their eggs in cornfields during August and September. The eggs overwinter and hatch the following spring in late May and June. If corn is present they feed and survive. The life cycle is broken by rotation as Northern and Western rootworms need corn to survive. Southern corn rootworms, on the other hand, overwinter in southern areas and fly north each year, laying eggs in the spring in planted cornfields. In some of the southern corn-producing areas, two generations a year may occur. After hatching, the larval form of the rootworm begins feeding and tunneling into roots. In severe cases corn may wilt and die from root pruning. Usually, however, the root pruning results in weakened stalks that are subject to lodging and yield reduction. Western and Northern corn rootworms are generally resistant to chlorinated hydrocarbons.

30. The influence of previous crops on the prevalence of soil insects in corn is as follows:

Underground corn insects	Other major factor	Year following meadow				
		1st	2d	3d	4th	5th
Wireworms	Soil moisture	XX	X			
Billbugs (3 species)		XX				
Cutworms (2 species)	Flooding, grass	XX	X	X	X	X
Sod webworms (5 species)		X				
Grape colaspis (2 species)		X				
White grubs (2-3 species)	Soybeans	XX	X			
Seed-corn maggot	Organic matter	XX	X	X	X	X
Cornfield ants		X	X	X	X	X
Corn root aphids		X	X	X	X	X
Southern corn rootworm		X	X	X	X	X
Northern and western corn rootworm			XX	XXX	XXX	XXX

31. Registered and effective alternatives to Aldrin for control of rootworms in corn are Furadan, Thimet, Dasanit, Dyfonate, Diazinon and Mocap. Counter has a temporary use permit and is expected to be registered for rootworms and wireworms before the 1975 crop year. Dow Chemical Company is presently seeking registration of Dursban. Insecticides which control resistant rootworm will also control nonresistant rootworm.

32. Diazinon is registered as a preplant control method for the cutworm and an application is pending for Furadan. Registered and effective insecticides for post emergent treatment are Carbaryl and Dylox baits or sprays and toxaphene sprays.

33. Registered and effective alternates to Aldrin for control of wireworms in corn are Dasanit, Diazinon, Dyfonate and Furadan. An application is pending for registration of Mocap. Thimet is labeled for reduction of wireworms.

34. No significant macroeconomic or microeconomic consequences will result from the suspension of aldrin for use on corn in 1975.

35. The Fuller Rose Beetle was recognized as a pest of Florida citrus in 1952 when large numbers were observed in several groves in Indian River and St. Lucie Counties. Since that time, this pest has been collected from 30 counties in the state. Its life cycle adheres to the 4-stage pattern inherent in beetles consisting of the egg, larvae, pupa, and adult. Eggs are deposited above ground, the hatchling larvae drop to the ground and enter the soil to feed for 10–11 months, as they mature, on roots. Pupation occurs in the soil and adults emerge from the soil to remain above ground feeding on the foliage, mating, and laying eggs. It is presently considered univoltine, producing but one generation per year. The adults feed on the young leaves of citrus and when in great numbers, cause serious setback of young plants. Adults also feed on the flowers and on rind of young fruit, resulting in unsightly peel scars when the fruit matures. Occasionally, young shoots may be devoured. The most serious injury by the pest is produced by the larvae which destroy the plant roots.

36. Affected trees have sparse foliage that may become chlorotic and wilt. When larvae are numerous, young plants may be killed in a short time or dwarfed. Older trees are more resistant, but do not grow well, are unthrifty in appearance, become poor yielders, and occasionally die. Since the damage caused by the larvae takes place underground, it often remains unnoticed until the plants start to wither and 'die back'.

37. Although the Fuller Rose Beetle has been collected from 30 counties in Florida, its economic significance is very circumscribed geographically. Of the 877,000 acres of citrus in Florida, the rose beetle is only present in numbers sufficient to commence to reduce yield on between 10,000 and 50,000 acres. The area of significant infestation is essentially the Indian River area of the Southeastern seaboard of Florida, an area characterized by poor internal soil drainage, a high water table, and consequently unusually shallow citrus root systems. A 1955 study indicated that in a typical Indian River grove, 75 percent of the feeder roots of citrus trees were located within 18 inches

of the top of the ridge of soil upon which citrus trees are usually planted in that area. Citrus trees won't extend their roots into waterlogged soils. The result is trees distinguishable by particularly restricted root systems with unusually limited supplies of feeder roots. These systems are less able to make do with decreases in root productivity resultant from insect damage which would be insignificant in other regions within the state.

38. Less than 5 percent of the total citrus acreage in Florida has ever been treated with any soil insecticide for control of any insect, and even within the Indian River Fuller Rose Beetle trouble region only 20 percent of the acreage has been so treated. The Fuller Rose Beetle is one of the more minor citrus pests in Florida. However, in some cases, the Fuller Rose Beetle is present in an area in such numbers that citrus yields are substantially reduced. In most of those instances, 2½ pound per acre treatments of aldrin twice during a growing season will provide adequate pest control. Citrus yields are reported to have markedly increased after insect damage and such treatment.

39. The theory behind aldrin/dieldrin soil treatment for citrus beetle control is that the chemicals should be incorporated in the surface of the soil surrounding citrus, creating a toxic barrier. Beetles may be killed during two stages of their development, when as larvae they drop from aerial regions of vegetation and enter the soil to feed, and when as adults they emerge from the soil to remain above ground, feeding, mating, and laying eggs. In a series of threshold tests in 1957 and 1958, aldrin provided approximately 78 percent control of rose beetles.

40. Aldrin/Dieldrin is overused on citrus to some extent, in the sense that it is unnecessarily utilized. Citrus growers can tolerate some crop loss before pesticide application is economically justified, yet before application of these chemicals they generally do not consciously formulate economic thresholds for determining when aldrin/dieldrin pays for itself in terms of insect control. In some instances, and particularly in the case of nurseries, these chemicals are employed as preventatives or insurance before insect damage is discerned. Many growers attempt to eradicate insect pests through applications of aldrin/dieldrin rather than reducing them to insignificant levels. In certain instances, however, the rose beetle substantially reduces crop yields absent the use of aldrin/dieldrin and without alternative means of control. In terms of the entire Florida citrus industry these instances are relatively rare.

41. The Coca-Cola Company, as one of Florida's largest citrus growers, does not use aldrin/dieldrin, receives fruit from groves located in areas where root weevil infestations occur, yet carries on profitable operations. The Company's decision not to utilize these chemicals was substantially the result of worker pressure resulting from possible health and safety problems involved in their use.

42. In view of the life cycle pattern of the rose beetle, whereby these insects generally mature from a larvae stage in the soil into adult weevils and then climb up weeds or citrus trunks or branches to lay their eggs, there is a large potential for disruption of the pest problems through cultural methods. If weeds and low-hanging citrus branches are cut down, major routes of access to the egg-laying areas of citrus will be closed off to the weevils. Particularly in California, certain sticky bands have been placed around trunks and have been effective in reducing the alternate path of weevil ascent. If the adult insects can effectively be denied such ascent, their damage to the aerial regions of citrus trees can be minimized and the insects procreative habits and efficiency can be stunted. Such means of pest control have not been extensively pursued in Florida.

43. California does not recommend the use of aldrin/dieldrin for control of the Fuller Rose Beetle on its very substantial citrus acreage although such insect also constitutes a pest of citrus in that state. Instead, the California spray program recommends malathion for control of the Fuller Rose Beetle, and both sevin and parathion to help with that beetle and to control certain other insect pests of citrus. Even within Florida, parathion and guthion, registered alternatives, are recommended as part of that state's spray and dust program. Various foliar sprays, most of which are already used in the Florida citrus program, some as often as 4 to 6 times a year, provide good initial kill of the adult weevil at issue. Included among these are malathion, furadan, sevin, guthion, orthene, lannate, supracide, and phosphamidon.

44. Suspension of the use of aldrin/dieldrin on citrus would not result in detrimental macroeconomic consequences. The need for treatment of the Fuller Rose Beetle is very confined, cultural and insecticidal alternatives are available and any adverse consequences will very easily become translated into a relatively minor shift in the supply-demand equilibrium. Nor are substantial microeconomic consequences anticipated.

45. No significant macroeconomic or microeconomic consequences will result from the suspension of aldrin or dieldrin until completion of the cancellation proceedings for all uses involved in these suspension proceedings in addition to corn and citrus.

CONCLUSIONS

I. Carcinogenic activity of a chemical can be detected by observation in man and by bioassay in experimental animals. The conclusive detection of the carcinogenic effect of a chemical by direct observation in man is extremely difficult. It may take 20, 30 or more years for a population to respond to a new chemical exposure with a significant increase of cancer cases due to the long latent period involved, that is, the time between exposure to a carcinogen and the manifestation of the effect, namely the tumor. In addition, the frequency of cancer in the population is very high, so that in order to demonstrate the existence of an increased risk related to a given exposure one needs a well-defined large population with known history of exposure and another comparable control population without that exposure. In the case of materials that become contaminants of the whole population, such as dieldrin,[5] this approach is almost impossible or nonapplicable.

Consequently, in the case of a food contaminant such as dieldrin where the identification of a non-exposed control population is difficult or impossible, the chances of detecting a carcinogenic effect by observations in man are extremely remote.[6] The human epidemiologic study by the Shell group of companies involving workers at the Pernis, Holland Plant[7] is admitted by the Shell Chemical Company not to be an adequate epidemiological study for cancer and was clearly so described by expert epidemiologists in these proceedings. In short, this study only examined a very small number of individuals for a period of time totally inadequate to assess a change in cancer risk extending over most of a lifetime.

For all practical purposes, the detection of carcinogenic activity of new chemicals is based on animal experimentation. All chemical substances or mixtures that have been proven carcinogenic by direct observation in man have also been shown to be carcinogenic in experimental animals with the exception of arsenic which is still under experimental study. Because of the difficulties of epidemiological studies on human carcinogenic exposures, there are usually no data which provide us with any evidence on whether cancer in man is caused by a chemical that has been shown to be carcinogenic in other mammalian species.

Bioassays are always performed on a number of animals which is extremely small when compared with the millions of humans exposed to most environmental carcinogens. Such studies can only detect carcinogenic effects resulting in fairly high incidences and the number of animals used in the tests is the main limiting factor of the sensitivity of the test system. The sensitivity of currently used animal bioassay systems is in most instances very limited. Therefore, any chemical which is detected as carcinogenic by such rather insensitive test systems represents a warning signal of great significance.[8] In fact, while it is customary or required that more than one species of laboratory animal be tested for carcinogenicity, a positive, confirmed finding as to one species is of extreme and grave importance.[9] This is reflected in the Delaney Clause or Amendment to the Federal Food, Drug, and Cosmetic Act (21 U.S.C. 348(c) (3) (A)) which provides that no food additive "shall be deemed to be safe if it is found to induce cancer when ingested by man or animal" and which "is generally intended to prohibit the use of any additives which under any conditions induce cancer in any strain of test animal." "Bell v. Goddard," 366 F.2d 177, 181 (7th Cir. 1966). Conversely, negative findings in carcinogenicity tests are of little significance in view of the insensitivity of the system.

II. Carcinogens are chemical, physical, or biological agents, exposure to which, of animals or humans, increases the probability of induction of tumors or neoplasia. This may be manifested by an increase in the number of individuals developing the tumor, an increase in the number of tumors in each individual, a decrease in the age at which the tumors appear, that is, reduction in the latent period of tumor induction, any combination of the above effects and perhaps the appearance of unique or unusual tumors.

---

[5] Surveys conducted by the Food and Drug Administration show that dieldrin is found in as much as 96 percent of all meat, fish, and poultry "composite samples" tested, and 85 percent of all dairy product "composite samples" tested. In addition, EPA surveys indicate that dieldrin is in approximately 90 percent of all air samples taken nationally and residues of dieldrin have been found in virtually *all* of the humans included in the EPA human monitoring survey. While the FDA surveillance program found less dieldrin present than in its market survey, the amounts found were still significant.

[6] The detection of the great cancer "epidemic" caused by cigarette smoking was made possible by the existence of a non-exposed population living in otherwise comparable conditions with those exposed. Also, besides the comparison of smokers and non-smokers, a quantitative estimate of the amount of cigarettes smoked make it possible to identify groups of population at different risks.

[7] Jager, Aldrin, Dieldrin, Endrin, and Telodrin: An Epidemiological and Toxicological Study of Long-Term Occupational Exposure (1970).

[8] It should be stated at this point, perhaps, that a relatively small number of chemicals, 700–800 or a maximum of 1,000, have proven to be carcinogenic in laboratory animals. It is not true that all or most substances can cause cancer in laboratory animals depending upon the dose applied.

[9] This is so, in part, due to the nature of cancer, that is, its irreversibility and long latency period following the initial exposure to the carcinogenic agent.

It is patent, it seems to us, that on the basis of our current knowledge or "conventional wisdom" the evidence is overwhelming that aldrin and dieldrin are carcinogens in the mouse.[10] This is established by the testimony of extremely well qualified and renown experts in the field of carcinogenesis such as Drs. Saffiotti, Heston, Farber, Epstein and others based on many laboratory tests of the mouse.[11] In fact, there are probably few pesticides whose carcinogenicity in mice is so thoroughly and conclusively documented.

This was, in effect, the conclusion also of the International Association for Research in Cancer which concluded in Volume 5, Monograph on the Evaluation of Carcinogenic Risk of Chemicals to Man, as follows:

> Dieldrin was tested by the oral route only in mice and rats. The hepatocarcinogenicity of dieldrin in the mouse has been demonstrated and confirmed in several experiments, and some of the liver cell tumors were found to metastasize. A dose-response effect has been demonstrated in both sexes with an increased incidence in females at the lowest dose tested, 0.1 ppm in the diet. (Corresponding to about 0.015 mg/kg bw/day). In mice there is no evidence of carcinogenicity in organs other than the liver.
>
> The available data in rats have not provided evidence of carcinogenicity at levels of up to 50 parts per million in the diet. (Corresponding to an intake of about 2.5 mg/kg bw/day).
>
> The experiments in dogs and monkeys were too limited in duration and/or group sizes to allow any conclusion to be made.

Further, witnesses for the Shell Chemical Company admitted at the hearing that the incidence of liver tumors in 5 different strains of mice evidenced statistically significant increases resulting from the oral dietary administration of dieldrin and many of the tumors in question have been diagnosed as unequivocally malignant.[12] The mice were of in-bred strains and an outbred and hybrid strain. The primary organ involved is the liver, but there was in addition a significant increase in tumors in the lung and other organs in some experiments. Further, positive dose-relationship in the incidence of liver tumors primarily and in lung and other tumors was manifest. Liver tumors metastasized to other organs within the animals and were successfully transplanted and, in at least some experiments, dieldrin shortened the latent period for tumor induction as well as increasing the incidence of tumors. Other evidence of dieldrin's carcinogenicity in the mouse is also present.[13]

The fact that dieldrin increased tumor incidence in mice of naturally occurring tumors does not alter our conclusions with respect to the findings in the mouse or their significance for man, to be discussed later in these Conclusions. As explained by Dr. Walter E. Heston, Chief of the Laboratory of Biology of the National Cancer Institute, a geneticist with 35 years in cancer research in experimental animals as a basis for the problem of cancer in man and the "father" of strains of test animals.

> A carcinogen, therefore, should not be defined only as something that produces tumors in a strain in which such tumors never occur without the carcinogen. Such a strain probably does not exist. A carcinogen is a substance that can increase the probability that a tumor will arise. It increases the incidence of a tumor in a strain and usually reduces that latent period of the tumor. In testing a substance for carcinogenicity, the aim, therefore, is to ascertain whether it can significantly increase the incidence of any tumor, and the choice of strain for demonstrating this is usually not the most susceptible, nor the most resistant but one with an intermediate genetic susceptibility.

In addition, Dr. Heston further testified that not all strains of mice or of any other species have the same incidence of spontaneous tumors and that "One cannot therefore state categorically that the mouse—i.e., all strains of the mouse—present an unacceptably high incidence of spontaneous tumors." As emphasized by Dr. Heston, well controlled experiments have been run with at least 5 strains of mice having different incidences of spontaneous liver tumors and it has been demonstrated from all strains that aldrin and dieldrin are carcinogenic in mice. Dr. Heston goes on to say that "Knowing this, and knowing the general biological similarity of mice and other mammalian species, including man, we can reasonably expect that in a population of human being exposed to Aldrin/Dieldrin, cancer of some kind will occur in some individuals, and that these individuals would not have been afflicted in the absence of these compounds."

The testimony and exhibits of the additional experts in carcinogenesis presented by respondent and the Environmental Defense Fund, Inc. convincingly support the view that the mouse is, indeed, an appropriate test animal for predictability to man. In short, most chemical carcinogens that have been adequately tested in different species show that they can produce tumors in all, or several of them. While the target organ may vary from species to species the concept of species specific carcinogens is not well supported. The mouse is probably the most widely utilized test animal, is the standard reference test animal in recently established and large scale programs of the United States Department of Health, Education and Welfare at the National Center for Toxicological Research for quantitation of toxicological and carcinogenic risk, and was extensively utilized, perhaps reluctantly, by the laboratory of the Shell organization at Tunstall, England.[14]

The following analysis by Dr. Umberto Saffiotti, Associate Director for Carcinogenesis, Division of Cancer Cause and Prevention, National Cancer Institute, a world renown expert whose initial testimony was cleared and approved by this organization and whose demeanor and knowledge during his several days of cross-examination especially impressed us, is helpful in this regard: [15]

> The argument that certain mouse liver carcinogens are "species specific" was recently reviewed in a paper by Tomatis et al.[16] entitled "The predictive value of mouse liver tumour induction in carcinogenicity testing—A literature survey." The authors searched the literature to make a list of chemicals that were reported to have induced liver tumors in mice: 58 chemicals were included in this list. The literature was then examined for reports on tests of these chemicals in two other species, rats and hamsters. Of these 58 mouse liver carcinogens, only 18 were reported to induce only liver tumors in mice, while the others produced also tumors in other organs. Of the 18 that were reported to produce only mouse liver tumors, none was reported to have been adequately tested in the other two species with negative results. Of the 58 chemicals which were reported to induce tumors of the liver, or of the liver plus other organs, in the mouse, only 16 were listed as having been tested and found negative in one of the other species (rats or hamsters); however, of these 16, 9 were reported as negative in rats but were not tested in hamsters, one was reported as negative in rats but was positive in hamsters, 5 were reported as negative in hamsters but were positive in rats. Thus only one compound, positive in mice, was reported as having been tested in both rats and hamsters with negative results: this compound is benzo[a]anthracene which not only causes hepatomas by feeding in mice, but also causes lung tumors, and was found to be carcinogenic also by other routes of administration in mice, causing tumors of the lung, skin and bladder. Although this compound was reported as negative in rats and hamsters, it is important to state that it

---

[10] Shell Chemical Company does not and, in reality, cannot dispute such conclusion. The position of Shell herein is, instead, to the effect, in part, that the mouse is not an appropriate animal in this connection, a contention we shall consider later in these Conclusions. The position of the Shell Chemical Company has been shifting on the issue of the mouse liver tumor and its significance and is also not in complete agreement with its witnesses. This makes it extremely difficult to prepare a decision in the very short period of time available, the preparation of which had to begin, therefore, prior to the filing of briefs or even the closing of the record, and may be prejudicial to the other parties. Consequently, this decision is responsive to what we had believed Shell's position to be and also to what it now is. We note, for example, that in its brief, Shell carefully avoids the word "cause" in connection with dieldrin and tumor incidence contrary to what was stated on the record of the hearing as to its position.

[11] While aldrin use accounts for nearly 95 percent of the total use of the 2 compounds, aldrin breaks down rapidly into its metabolite dieldrin. Consequently, residues found in man and the environment are principally dieldrin residues and thus the hazards of dieldrin are of prime significance.

[12] There is no valid distinction between the induction of benign or malignant tumors in determining the carcinogenicity of a compound and Shell Chemical Company and its pathologist witness employed at Tunstall do not contend that there is although some of the cancer experts testifying on behalf of Shell appear to make such distinction.

[13] The evidence in these consolidated suspension proceedings went beyond the evidence available to and the conclusions of the IARC quoted above. In addition, our conclusions are not affected by the last minute revised data differing from prior published studies adduced by the Shell Chemical Company. Also, time is lacking for an analysis of each of the mouse experiments involved and no useful purpose would be served thereby.

[14] Also significant is the fact that an experimental study involving approximately 25,000 mice, was established using a carcinogen which is known to produce liver cell tumors in mice as well as a variety of other tumor types in mice and in other species.

[15] In fact, much of the preceding section of these Conclusions was based on the testimony of Dr. Saffiotti, confirmed and corroborated by the testimony of many other cancer expert witnesses.

was not adequately tested in rats and hamsters at all. There are no reports of such tests by chronic feeding in rats or hamsters, nor of any long-term tests with continuous administration in large numbers of animals, with adequate pathology. The only feeding study in rats on this compound was published in 1945 [illegible]; it states that 2 out of 3 male rats were found to have 3 hepatomas each. No hepatomas were found in 3 females, nor in different groups of controls. Although inadequate, this report suggests the possibility of liver carcinogenicity in rats. So the conclusion is that no chemical was found to have been adequately tested and shown to produce liver tumors in mice but no tumors in the other two most common species of test animals. As a matter of interest, Tomatis et al. have limited their discussion to the correlation of test results as presented in the literature, without any critical evaluation of the adequacy of the tests used to enter a classification of positive or negative into their tables. Such an analysis would show that many tests in rats or hamsters, reported as negative, are really quite inadequate and should be rejected as "negative evidence."

The survey by Tomatis et al. is, however, sufficient to disprove the proposition that the induction of liver tumors in mice is a tissue response that is not representative of carcinogenic effects such as are seen in other organs or other species. A few people have proposed that the carcinogenic response of mice is not representative of that of other species including man. No scientific basis could be found to support this argument.[16]

The Report of the 1973 Joint Meeting of the FAO Working Party of Experts on Pesticide Residues and the WHO Expert Committee on Pesticide Residues, which is not the official view of WHO but only that of the participants of the expert committee, stated, in part, as follows:

> * * * The Meeting agreed that there is a serious lack of knowledge regarding the processes involved in the development of liver tumors by mice and that it would be unwise to classify a substance as a carcinogen solely on the basis of evidence of an increased incidence of tumors of a kind that may occur spontaneously with such a high frequency.
>
> In general it was felt that if the exposure of mice to a pesticide was associated with an increased risk of the development of liver tumors, long-term feeding studies on at least one other species should be required. Carcinogenicity tests in two species other than the mouse would be regarded as appropriate where it was evident that man might be exposed through food to a dose level close to one that increased the incidence of liver tumor in mice.
>
> The meeting agreed that, although the above considerations might be useful for general guidance, it would be essential for each pesticide to be considered and assessed individually.

This does not detract from the testimony of Drs. Heston, Saffiotti and others with respect to the significance of mouse liver tumors. The FAO/WHO report recognizes that the matters there stated "might be useful for general guidance" but that each pesticide should be considered and assessed individually. It appears to us that the quoted material set out above from the FAO/WHO report is basically the view of Dr. Roe who testified on behalf of Shell Chemical Company herein and who was one of the few or, perhaps, 2 cancer experts on the expert committee. He admitted at the hearing, in effect, that the members of the expert committee can determine the report that is issued. For the reasons stated herein for, in effect, giving little weight to Dr. Roe's testimony in this connection, we similarly so regard the FAO/WHO report.[17] We just do not believe, on the basis of this record, that it represents the current state of our knowledge or the accepted scientific view. We are, instead, impressed by positive findings in 5 different strains of mice with differing incidences of spontaneous tumors. As we stated above, inbred, outbred and hybrid mice were involved in the experiments. (See also discussion which follows on other tumors of the mouse, and the rat). Moreover, Shell's own experiments clearly demonstrate how natural variability can be surmounted and an unequivocal result be obtained. For example, from a consideration of the frequencies of malignant hepatic neoplasms, as diagnosed by Shell's pathologists, it is apparent their spontaneous incidence in control animals is neither high nor variable, while the dieldrin treated groups consistently show marked and often high incidence of such malignancies.

Shell Chemical Company further contends that a large variety of factors, chemical and nonchemical, can greatly alter the incidence of tumors in the liver in the mouse and, thereby, challenges the appropriateness of the mouse as a test animal and its applicability to man. Specifically, Shell has reference to the fact that sex, hormones, diet and other factors can influence the occurrence of cancer in test species. This is well known to cancer investigators and we believe the following answer by Dr. Heston to the matters raised by Shell witnesses disposes of some of the contentions of Shell's witnesses in this regard:[18]

> * * * Besides those noted by Dr. Roe, there are probably many other factors, as yet undiscovered, which can affect the incidence of tumors, and this likelihood applies not only to hepatomas, but also to other tumors as well. And, given a fundamental biological similarity between the mouse and other test species, it is obvious that many of the factors cited by Dr. Roe and others as influencing the incidence of tumor formation in the mouse would have a similar effect on other species as well. It is merely because we have studied the mouse in greater detail than other species that there is a greater literature concerning spontaneous tumors in the mouse than in other test animals.
>
> All of Dr. Roe's discussion of factors affecting tumor incidence, however, has absolutely no bearing on the question of carcinogenicity. Most simply put, the question is "Can the administration of Aldrin/Dieldrin to test animals result in some of their cells becoming malignant?"
>
> This question is answered by selecting two groups of test animals which have been bred under the same conditions and which have similar genetic characteristics. Both groups should be alike with respect to sex; both groups should be tested at the same time in identical surroundings; both should be given the same nutrition. In all respects except one, in short, the animals of both groups should exist under the same conditions. The only difference is that on one or more occasions, one group will be exposed to a known quantity of the compound under test and the other will not.
>
> Thereafter the incidence of tumor formation and other data will be noted, and through statistical analysis one can determine whether any increased incidence of tumors has occurred in exposed animals when compared to controls. If so, and if the difference in incidence is sufficiently great, we can reasonably attribute the increased incidence to exposure to the compound under test. We do not thereby conclusively prove that the test compound "caused" the elevated incidence, as Drs. Roe, Sternberg, Newberne and others would require; if we had to prove causation we could not establish any substance as carcinogenic even today. Rather we must and do make judgments as to carcinogenicity on the basis of statistically-significant differences in tumor incidence arising from valid experiments such as I have outlined above, and from other information at hand.
>
> Whether the particular strain or species of test animal chosen has a high, medium or low incidence of spontaneous tumors is therefore irrelevant so long as animals are assigned without bias to test and control groups. The fact that diet can increase or decrease the incidence of tumors becomes irrelevant to long as both exposed and control animals are fed the same diet. All of the other factors cited by Dr. Roe and others similarly are irrelevant so long as they apply equally to control and exposed test animals.
>
> Does the variability in the incidence of spontaneous tumors in the mouse make it an inappropriate animal for carcinogenicity testing? Do any of the other factors cited by Drs. Newberne, Roe, Stevenson and Thorpe lessen the value of the mouse in determining possible carcinogenic threats to human health? For the reasons I have given above, the answer is an emphatic no.

---

[16] It should be pointed out at this point that the Tomatis article further stated that "The present review indicates that the induction of liver tumors in the mouse should be considered as valid as the evidence obtained in the rat and/or the hamster at any site. It does not imply that the chemical which has been tested with negative results in one or more species should be automatically regarded as having a possible carcinogenic effect on man solely on the grounds that it induces liver tumors in the mouse. Conversely neither does it imply that negative results in the mouse must be regarded as proof of safety." Aldrin and dieldrin have not on the basis of adequately conducted and reported experiments at proper dose levels been tested with negative results in the rat and do not appear to have been tested in the hamster at all.

[17] Similarly, the only cancer expert on the Administrator's advisory committee was introduced as a witness for the Shell Chemical Company, and we feel that the record herein totally overcomes his testimony with respect to the significance of mouse liver tumors and the standard by which cancer risk to man is determined.

[18] It should be noted that the Shell employee witness with overall scientific responsibility for the toxicology programs in Shell's Tunstall laboratories testified that the laboratory tried to eliminate environmental biases as much as possible in the various mouse tests on the carcinogenicity of dieldrin and the record does not indicate any such biases in the mouse tests involved. Also, Dr. Heston's testimony set forth above with respect to the irrelevancy of the matters raised by Shell was echoed by other cancer experts herein. Further, variability in spontaneous tumor incidence is found not only in the mouse, but also in other species *including man.*

Reproduced from best available copy.

In this connection, it is helpful to set forth detail some of the testimony of Dr. Arthur J. Upton, Dean, School of Basic Health Sciences, State University of New York at Stony Brook, New York, a noted cancer expert. He states as follows: [19]

The emphasis by Shell witnesses that knowledge of mechanisms must be defined before any agent can be considered carcinogenic, even though this agent has been demonstrated to induce carcinogenic effects in valid experimental systems, can only be regarded as misleading in extreme. In fact, in spite of a very considerable amount of research, the basic mechanisms of action of any single carcinogen have not yet been elucidated. This requirement of Shell would define away the entire field of chemical carcinogenesis.

It should be noted that the Delaney Amendment does not utilize the word "cause," but, instead, deals with *food* additives which "induce" cancer.

I would like to turn now to a discussion of the basis on which findings of carcinogenicity are made in animal experimentation. In particular, I would like to address the following argument: Even if an increased incidence of tumors is found in test animals after exposure to a particular compound, one cannot properly assert that the test agent "caused" the induction of such tumors; one can state only that a statistical association was demonstrated between administration of the compound and the elevated incidence of tumors. One must know the mechanisms by which a carcinogenic response is elicited before one can speak to the question of "causation" or label a test compound a "carcinogen".

I do not subscribe to this position. In carcinogenicity testing today we base findings of carcinogenicity on precisely these statistical associations that have been described above as inadequate, and I believe it not only proper but important that we do so. Given our present state of knowledge concerning the mechanisms of carcinogenicity, it may be some time before we can reliably establish the entire pathway from administration of a carcinogenic agent to the elicitation of a carcinogenic response. To require that such a pathway be established in detail before an agent can be labelled "carcinogenic" would be to adopt the ostrich-like position of ignoring facts which constitute obvious warning flags for human health.

A foremost reason why we cannot wait for a full explanation of mechanisms of carcinogenesis is because of their apparent multiplicity and complexity. It is no longer reasonable to assume that cancer results from a single factor; rather it appears that carcinogenesis is a multi-causal, multi-phased process in which genetic, hormonal, environmental, and other factors play varying roles in the elicitation of a particular carcinogenic response. At this stage of our knowledge it is true that we can make some generalizations concerning particular factors. We can say, for instance, that mammalian neonates appear to be more susceptible to the actions of some carcinogens than older animals; but even here one should note that the relationship of age to tumor incidence appears to vary with the type of tumor in most species studied. In man, for instance, some forms of cancer appear predominantly among children, while others seldom appear among the young yet increase exponentially with age in adults. It is because of these and other sharply differing patterns of cancer incidence in man and other mammalian species that the process of carcinogenesis appears to involve a large number of variables and highly complex series of interactions. Hence it is doubtful that we will understand fully the mechanisms of even the simplest forms of carcinogenesis in the immediate future.

Because of incomplete knowledge concerning mechanisms, I also do not believe that distinctions between "carcinogens" and "co-carcinogens", or between "causative agents" and "enhancing agents" can be considered relevant today when ascertaining hazards to human health arising from carcinogens. In safety testing of carcinogens today we are concerned with one question: "Does exposure to the test agent result in a significant induction of tumors in exposed populations as compared to controls?" If so, then the test agent has elicited a carcinogenic response and must therefore be considered potentially hazardous to human health. Whether the agent actually is a *sine qua non* of the observed response or merely enhances a virus or some other factor found in the host animal is irrelevant unless and until we know that similar factors are not also found in man. Until we have such knowledge, we have no basis on which to make distinctions between "carcinogens" and "co-carcinogens" and "causative agents" versus "enhancing agents".

Given this lack of knowledge concerning mechanisms, I believe that a carcinogenic reaction in any species of test animal must be considered sufficient to describe the test compound as a carcinogen and so a threat to human health. I consider that a similar reaction in a second mammalian species is a confirmation of the carcinogenicity of the test agent, but it is not necessary before a finding of carcinogenicity and threat to human health can be made; and negative results in a second or even third species of test animal do not in my mind establish that the test agent is not a threat for human beings. Given the variation in human susceptibility to carcinogens, I believe it unreasonable to ignore a finding of carcinogenicity in any mammalian test species when considering possible effects on human health.[20]

We have limited our considerations above with respect to the carcinogenicity of dieldrin to the results in the mouse and specifically in the mouse liver. We think it is clearly a carcinogen solely on that basis. (See also Part III of these Conclusions). But, we are not restricted by the record solely to that organ in the mouse or solely to that test animal. While the effects of dieldrin were manifested primarily in the liver of the mouse, there was also statistically significant increases of tumors in the lung and other organs of the mouse in some of the experiments as published and also with the newly introduced but questionable revised data. Even with the revised data it is clear that dieldrin at low feeding levels, at either 0.1 ppm or both the 0.1 and 1 ppm levels, can elevate the incidence of tumors at sites other than the liver and that this elevation is highly significant in either males or females or in both sexes, as demonstrated by Dr. Gross, a well qualified statistician and cancer expert, by conventional and accepted statistical analysis. These findings tend to corroborate the carcinogenicity of dieldrin in the mouse,' as evidenced by the reaction of the mouse liver to dieldrin, the applicability of that finding to man and to weaken Shell's arguments based exclusively on the liver of the mouse.

Also, there is experience with the rat. We are hesitantly unwilling at this time to find that dieldrin is conclusively a carcinogen in the rat although there are indications that this is so especially when the chemical is tested at the lower dosages. This is the case, we believe, because of the effect of competing toxicity at the higher feeding levels. It can and should be stated in this connection, however, that while we are uncertain with respect to our failure to find that dieldrin is a carcinogen in the rat, we are certain, nevertheless, that the findings in the rat cannot be described as negative.

III. Also in connection with the mouse and its significance for man, Shell Chemical Company contends that phenobarbital, an alleged dieldrin-imitating enzyme inducer in the mouse liver, does not cause cancer in man to illustrate, thereby, the inapplicability of mouse liver tumors for man. Specifically, Shell states that * * * phenobarbital is a dieldrin-simulator in the mouse; it acts the same way as does dieldrin in increasing the incidence of mouse liver tumors. Phenobarbital does not cause cancer in human beings, even though it produces a tumorigenic response in the mouse liver. This shows that the mouse in this respect is a highly inappropriate test animal with which to make a judgment as to human carcinogenicity."

Dr. J. Clemmesen of the Danish Cancerregisteret and the author of a recently published paper entitled "Are anticonvulsants oncogenic?" was presented by Shell Chemical Company in an effort to show that agents, such as phenobarbitone, which can cause certain kinds of enzyme changes and which are carcinogenic in some animal systems, are not carcinogenic in man. This is contended by Shell to provide an example of a substance carcinogenic in the mouse but not in man.[21]

The paper deals with the experience of a group of epileptics who received a regular treatment of sedative drugs, including phenobarbitone. The roster of the epileptics at Filadelfia, a Danish epileptic hospital, was compared with the roster in the Danish National Cancer Registry to see how many of these people had developed any form of cancer in the course of their treatment for epilepsy.

We cannot agree with Dr. Clemmesen that his paper or study establishes that phenobarbitone is not carcinogenic in man. Over 80 percent of the patients at Filadelfia were admitted at an age under 40 years and approximately only 23 percent of the patients survived 20 years of treatment. In fact, 42 percent of the male and 39 percent of the female patients were under 20 years at time of admission. It appears to us that the percentage of patients who reached the cancer-susceptible advanced ages after any reasonable duration of treatment must be small and that these patients are apparently dying of competing causes before cancer develops or are still too young to develop many cancers. Although the study was age adjusted, the participants therein or subjects thereof were too young for meaningful or conclusive analysis. This is also the view of Dr. Marvin A. Schneiderman, Associate Director for Field Studies and Statistics, Division of Cancer Cause and Prevention, National Institute, a well qualified biometrician. He further concludes that "the data here are consistent with the possibility that the anticonvulsants which the epileptics received increased the risk of liver cancer, perhaps two or three-fold. Thus, in the case of phenobarbitone, the mouse may indeed b[y] an appropriate model for human carcinogenesis." Dr. Schneiderman lists many other reasons for his similar disagreement with Dr. Clemmesen. In short, we do not believe that the non-carcinogenicity of phenobarbital to man has been established on the basis of Dr. Clemmesen's paper. Shell's argument bottomed thereon must fall.

---

[19] See also testimony of Dr. Samuel Epstein in the consolidated suspension proceedings (EDF Exhibit No. S 2). As part thereof he stated:

[20] In addition, on cross-examination this witness indicated that matters such as casein and diet which affect tumor incidence in the mouse could conceivably be similarly carcinogenic in man under certain conditions. On the basis of our current knowledge, we clearly cannot state with certainty that the factors cited by Shell as influencing the occurrence of liver tumors in the mouse cannot similarly increase tumor incidence in man in the liver or elsewhere.

[21] There is current additional inquiry as to whether anticonvulsants are carcinogenic in man. It should also be stated that Dr. Clemmesen's study could only be considered as pertinent to the dieldrin carcinogenesis problem in a peripheral way and the results thereof could not necessarily be extended to dieldrin.

While the Shell Chemical Company agrees that the Jager study, as supplemented by additional data, of the workers at the Pernis, Holland plant could not be taken as statistical proof that dieldrin is not carcinogenic to man, it contends that the absence of "premonitory" or precancer signs in the workers is positive evidence against the possibility that dieldrin is carcinogenic to man. Shell emphasizes that it is unusual for primary liver cancer to develop in man without premonitory signs such as liver injury, enzyme induction and detectable alpha-feto protein. The Jager study has probative value in the direction advanced by Shell, but it clearly does not establish that dieldrin is not carcinogenic in man or that the mice study results are inappropriate.

While it is expected that dieldrin would manifest itself in the human liver, this is not necessarily so. Consequently, the normal liver function of the Pernis workers does not establish absence of carcinogenic activity. In addition, Dr. Farber, Director of the Fels Research Institute, Temple University School of Medicine, who has expertise with respect to animal and the human liver, testified that cancer of the liver could develop even absent "premonitory" signs or in the face of normal liver function "until perhaps late in the course of the disease." He explained that if the patient had cirrhosis of the liver which is a chronic disease of the liver which frequently accompanies and precedes liver cancer, then functional changes would be manifest, but that cancer can develop in the absence of cirrhosis and such individuals may not have disturbances in the liver function until late in the course of the disease. Also, the presence of alpha-feto protein in the blood serum is not necessarily found in liver cancer patients.

Moreover, the working population at Pernis was screened by medical examinations before employment and had further examinations during the course of employment. Workers with abnormalities of the brain and liver, who might be most susceptible to dieldrin effects, were excluded from the study at the start thereof. Persons who showed signs of insecticide intoxication or who themselves were distressed by personal reactions to the insecticides were shifted away from direct exposure. Shell should be commended for such action. But, it resulted in a selected population of relatively healthy young male industrial workers. In this connection, Dr. Schneiderman concluded that "the Jager study is an interesting followup of some healthy young male workers on whom we have only rudimentary dose information, but who appear to have received relative small doses of the material, and who have been followed for relatively short periods of time with no overwhelmingly destructive effects yet appearing." While this observation or conclusion was given basically from an epidemiological viewpoint, it also applies to the absence of "premonitory" signs, we believe.=

Shell Chemical Company further states that approximately 1,000 workers have been exposed to dieldrin and other pesticides at the Pernis plant and that if dieldrin were a human carcinogen this could very well have been detected in a group of this size. Shell contends that "virtually all known human carcinogens have been observed first in small industrial populations, most with numbers smaller than the worker population at Pernis."

The figure of 1,000 workers is not valid as extended exposure and surveillance did not cover a group any where near that size. But, in reality, Shell is engaging in an argument involving epidemiology, a matter which it allegedly conceded. More importantly, Shell's contentions in this regard are based upon the testimony of Dr. Van Raalte with respect to 18th, 19th and early 20th century discoveries of cancer in small groups of workers and the inferences he drew therefrom. Such testimony and inferences were totally negatived we believe by Dr. Schneiderman's distinction between retrospective studies in an epidemiological sense and prospective studies. He stated, in part, "If you are doing a retrospective study, that is you take people with the disease and go back then and try to see what they worked on, you can find very much more in retrospective study than in prospective study. Almost all the ones you have talked about are retrospective study. * * * Now here the Pernis study is a prospective study. It deals with somewhere up to 800 men who were followed through the future. We are not looking at people with liver cancer to see where they worked. We are looking at people who worked to see whether they develop some disease."

Shell Chemical Company goes on to make what we believe are, in part, epidemiological arguments, that is, period of exposure, latency periods, level of exposure, the worker population at issue and their relation to the general population and the significance of the absence of women and children from the worker group. It seems to us that Shell cannot on the one hand state that it does not contend that the Pernis study was epidemiological proof of the negative and rely on the matters listed above as it does. Comparisons with experiences with known human carcinogens in the respects listed above, while of borderline relevancy, does not necessarily tell us anything about dieldrin.= Shell concludes, in effect, with the statement that "were dieldrin a human carcinogen, the results at Pernis would have been different." Such is clearly not the case. All that can be said with respect to the Pernis experience at his time is that an excess of cancers has not yet appeared in those workers.

Alleged similarities or dissimilarities between the mouse, man and other species were also advanced by Shell Chemical Company. Dr. Wright, a Shell employee, described the process of degranulation of the rough endoplasmic reticulum and stated that degranulation was elicited by dieldrin within the liver cells of mice, but not of other species. He testified that this process was closely correlated with carcinogenicity and opined that this would prove to be a critical process in carcinogenesis and would soon provide a predictive test.

However, Dr. Farber pointed out 2 known exceptions to the correlation advanced by Wright, which compounds are positive for carcinogenicity in the rat but negative for degranulation in the rat. Also, dieldrin is negative for degranulation in the male LAGG strain mouse, but positive for carcinogenicity. Likewise aflatoxin B, negative for degranulation in human liver cells is at least strongly suspected of carcinogenicity in man.

An *in vitro* degranulation test as a valid index for carcinogenicity is not established or accepted or anywhere near acceptance in the scientific community and is, in fact, a theory lacking in conclusive proof and already subject to exceptions. Dr. Wright has confined his work up to this point to one or two species and the contentions of his employer in these proceedings on the basis of his work is speculation based on limited knowledge. Even if the correlations advanced by Shell might shed some light on one of the interactions which take place in the carcinogenic process, we would still be far from an explanation of that process in any single species to say nothing of an explanation of how various species compare and contrast among themselves in their reaction to carcinogens. In short, even if degranulation should correlate with cancer incidence, this phenomenon may tell us nothing concerning mechanisms, much less shed light on the differences among species insofar as causative mechanisms are concerned.

Dr. Wright also suggested that the induction of microsome enzymes in the mouse liver was closely associated with carcinogenicity. This is an alleged association based on only 5 compounds and at least one exception thereto is known. Dr. Farber was emphatic in rejecting a precise correlation between enzyme induction and carcinogenicity. But, what we do know of enzyme induction by dieldrin is not reassuring. Dr. Gelboin of the National Cancer Institute, who discussed in detail the liver microsomal enzyme system, compared it to a "double edged sword." Stimulation of microsomal enzymes by foreign chemicals serves an important function in enabling the body to more rapidly detoxify and excrete toxic chemicals. But, it is now known that in certain circumstances, microsomal enzymes activate carcinogens by converting them to their active forms. Enzyme induction in itself thus conveys a warning of possible carcinogenic hazard, not only to animals but also to man. Dieldrin induces liver enzymes in rats as well as mice and there is evidence that it also acts in man. The alleged "no effect" level for enzyme induction by dieldrin in man in the Pernis study has been countered by a later study, using a different assay, which showed elevated enzyme activity associated with relatively modest blood levels of dieldrin.

In addition, Dr. Farber testified that, "It is evident that many chemicals require metabolic conversion to active derivatives before they can initiate the development of cancer. However, the specifics of the metabolic processes which result in cancer in various test

---

= In this connection, Shell strenuously contends in its brief that the malignant tumors suffered by 2 of the Pernis workers cannot be related to the workers' exposure to dieldrin and to the other chemical compounds manufactured there. We do not hereby conclude that there is any such connection. But, we do not believe that Shell can establish that there is not taking into account the variable sensitivity of humans to carcinogens and the fact that the cancer may manifest itself in different organs.

= Much was said about vinyl chloride. It is not comparable to dieldrin and the Pernis workers, and we note that its carcinogenicity was discovered in laboratory animal experiments.

animals are not clear, to say nothing of the metabolic processes in man. No one as yet can draw any valid correlation between a particular pattern of metabolism and the induction of cancer in any species, and any judgments concerning carcinogenicity or lack thereof based on metabolic patterns have no scientific basis at this time." This observation relates to the testimony of Dr. Hutson adduced by Shell with respect, in part, to the rate of metabolism and carcinogenesis. As observed by Dr. Farber, "Suffice it to say that while metabolic activation is essential to carcinogenesis, no correlation between the degree of metabolic activation and carcinogenic risk has been established by anyone for any compound tested in any species to date."[34]

IV. In the absence of conclusive evidence derived from studies in man for either the safety or the carcinogenicity of aldrin/dieldrin, we are forced to make a judgment as to the potential hazard posed by dieldrin to man on the basis of experiments with animals. The scientific community has accepted the results of laboratory experiments with rodents as an indication whether chemical agents are likely to be carcinogenic in man, as has the Congress as reflected in the Delaney Amendment. Reliance upon animal studies is possible primarily because the pathological processes of tumor development in man are very similar to those of other mammalian species.

Reliance upon animal studies is supported by experience as well as by the pathological similarities of man and animals. Many chemicals which are known or suspected to be carcinogenic in man were first identified as carcinogens in mice. These include coal and tobacco tar extracts, polycyclic and heterocyclic aromatic hydrocarbons, estrogens, and carbon tetrachloride. Furthermore, as stated earlier, all chemicals which are known to cause cancer in man also have been shown to produce cancer in laboratory animals, with the possible exception of trivalent inorganic arsenic which is still under study.

This is not to say that the biological processes of mice and men are identical in every respect. Chemical carcinogens, for example, may affect different target organs in different species. Generally, however, there are sufficient similarities in the metabolic and biologic processes of experimental animals and man to indicate that an agent causing cancer in rodents or other experimental animals poses a high risk of causing cancer in man.[35]

The record is replete with evidence, in fact, overwhelmed with evidence, some of which has been set out above, that such is the case here. We believe that this conclusion represents established traditional and "conventional wisdom." The Shell Chemical Company has strenuously and with sophistication attempted to demonstrate that "this truth" does not apply to aldrin and dieldrin for the reasons we have detailed above. We do not believe that traditional wisdom or science has been overcome thereby. Shell's presentation with respect to the shortcomings of the mouse as an appropriate test animal and its lack of significance for man is based, in part, on matters far from established in the scientific community, speculation and surmise. In reality, our knowledge with respect to cancer is very limited. Many, many years would be required to pursue the theories, hypotheses and correlations advanced by witnesses for Shell without any confidence that they could be proven.

We find, on the basis of the considerable record herein, as discussed above in part, that aldrin/dieldrin pose a high risk of causing cancer in man. We believe that the respondent, who has the burden of going forward to present an affirmative case for suspension, but not the ultimate burden of persuasion as to safety,[36] has in fact satisfied the burden of proof which is not his that the chemicals in issue pose a high risk of causing cancer in man. It is true that we cannot now point to any individual as having cancer caused by these chemicals, but we may not be able to do so even if aldrin/dieldrin were established human carcinogens due to the many other substances or chemicals in man's environment and the absence of a control population. We cannot wait to do so, however. It would be irresponsible in the extreme to pursue such course or to insist on knowledge of the mechanisms of cancer before any test agent can be regarded as carcinogenic.

The issue of carcinogenicity of aldrin/dieldrin assumes extraordinary significance and immediacy in view of the fact that the entire population of the United States is continually exposed to these chemicals and that dieldrin has probably accumulated in the body tissue of almost every individual. Dieldrin is stored in human fat, circulated in the blood, transferred across the placenta to developing fetuses and secreted in human milk. Dieldrin is a persistent chemical which pervades our diets at significant residue levels. Additionally, man is exposed via the air and other routes. No useful purpose would be served and time does not permit the listing in great detail[37] of the quantities and extent of dieldrin found in humans, in human maternal milk, and in foods or describing in detail the fact that the agricultural uses of aldrin and dieldrin result in much of the dietary exposure of dieldrin to man. It is sufficient to state that dieldrin is found in substantial amounts in humans and in our diets and that a significant source of that dieldrin is the agricultural uses at issue. In addition, there are no indications of a consistent upward or downward trend in residues in human tissues or food.

Averages are usually cited in connection with the amount of dieldrin found in humans, ingested in our foods, etc. We have often stated that we have special concern with the persons with the highest body burdens of dieldrin or persons who take in the highest amount of dieldrin in their diets, etc. We are also especially concerned with those in the population who are genetically the most susceptible and the very young. It is essential, we believe, that the cancer hazard of dieldrin be viewed with this perspective.

V. Shell Chemical Company makes several arguments based upon the announcement at the hearing that heptachlor and chlordane, which apparently may contain heptachlor, will be available for use on corn in 1975, that is, will not be the subject of suspension proceedings under the act. Shell states that heptachlor and its major metabolite, heptachlor epoxide, increase the incidence of tumors in the mouse to the same extent as dieldrin and that the failure to suspend heptachlor and chlordane indicates that "the Environmental Protection Agency does not contend or believe that a compound presents an 'imminent hazard' on the basis of a 'tumorigenic response such as that found in dieldrin-treated mice. The Agency requires more than the mouse."

In his Determination and Order of December 7, 1972, in the consolidated aldrin-dieldrin cancellation proceedings, the prior Administrator, in deciding not to suspend such insecticides stated, in part, that "the present evidence, confined to one strain of mouse is tentative evidence of a 'risk,' but not sufficient proof that aldrin/dieldrin is a carcinogen in human beings. If unrebutted, this evidence would be a caution signal as to long-term exposure, but does not amount to a red light requiring immediate elimination of all dieldrin residues in the diet." The situation with respect to heptachlor and heptachlor epoxide is similar to that stated for dieldrin by the Administrator on December 7, 1972. As far as we can determine, there is one mouse experiment which incriminates this chemical.

We are somewhat surprised by Shell's position in this regard. It has constantly required and demanded reproducibility of results and confirmation of findings. Its case is bottomed, in part, on these requirements. There is no established confirmation or reproducibility with respect to heptachlor and we do not find any discrimination or capriciousness by virtue of the failure to suspend. Consequently, also, we cannot agree with or follow Shell's argument that the banning of aldrin/dieldrin will not prevent an imminent hazard due to its replacement only in limited party by heptachlor and chlordane. In short, certainty as to the carcinogenicity of heptachlor has not, by any means, reached the level of certainty as with respect to aldrin/dieldrin.

Shell makes additional arguments in connection with the effects of the failure to suspend heptachlor and chlordane. In the consolidated aldrin/dieldrin cancellation proceedings the respondent did not advance these 2 chemicals as proposed alternatives to aldrin/dieldrin or as alternatives that it would sponsor and defend. This fact had nothing to do with the actual availability of these insecticides as alternatives to aldrin/dieldrin. It was a position taken in that proceeding so that the chlorinated hydrocarbons could not be cancelled in turn on the basis that there was another one to take its place and respondent did not, and in actuality now does not, sponsor such chemicals as substitutes for aldrin and dieldrin. Shell contends that "By Respondent's late reversal of its position with respect to heptachlor and chlordane availability and use as a *de facto* alternative to aldrin in 1975, Shell has been severely prejudiced in presentation of its case" in violation of the notice requirements of 5 U.S.C. 554(b). We find no merit to such argument.[28]

---

[34] At the oral argument herein at the close of the hearing, Shell Chemical Company set forth for the first time a 5 stage scheme for dieldrin induced tumor development in the mouse liver and contended that 4 of those stages were only found in the mouse. Such is not the case.

[35] Dr. Heston, a noted geneticist with much experience, testified as follows in this regard: The human population is so much more genetically diverse than any laboratory animals that if a chemical has been shown to be carcinogenic by a significant induction of any kind of tumors in any laboratory strain of mammal, we can reasonably expect that at least certain human beings would also respond to the chemical by developing some kind of neoplasm.

[36] See section 164.121(g) of the rules of practice. See also e.g., Stearns Electric Paste Company v Environmental Protection Agency, 461 F. 2d 293 (7th Cir. 1972); Continental Chemiste Corporation v Ruckelshaus, 461 F. 2d 331 (7th Cir. 1972); Environmental Defense Fund, Inc. v. Ruckelshaus, 439 F. 2d 584 (D.C. Cir. 1971).

[37] It should be stated briefly that dieldrin is widespread in human food throughout the United States. It occurs most frequently and in greatest quantities in foods of animal origin, that is, dairy products, meat, fish and poultry. Dairy products are probably responsible for the greatest contribution of the average dietary intake but the most highly contaminated single food group is fish. Residues of dieldrin in dairy products are especially high in the Corn Belt and neighboring states. The FDA Market Basket Survey provides a misleading low estimate of average dietary intake of dieldrin. Persons with high dietary intakes of dairy products and meat, especially children, have higher daily intakes than average, often much higher. Breast-fed infants have the highest daily intakes of all.

The additional arguments advanced by Shell Chemical Company in connection with the availability of heptachlor and chlordane in 1975 are all bottomed on the premise that "the evidence demonstrates that heptachlor-heptachlor epoxide is as much, or more, a laboratory carcinogen as aldrin-dieldrin." This is not the case at this time. Those arguments must fail.

VI. As stated earlier, our consideration of "imminent hazard" with respect to aldrin and dieldrin must take into account "the economic, social, and environmental costs and benefits" of these pesticides. In other words, even with respect to "imminent hazard" a risk-benefit analysis is required by the statute. CF "e.g., In re Stevens Industries, Inc.," 2 E.L.R. 30011 (June 2, 1972), affirmed "Environmental Defense Fund, Inc. v. Environmental Protection Agency," 489 F. 2d 1247 (D.C. Cir. 1973); "Environmental Defense Fund, Inc. v. Ruckelshaus," 439 F. 2d 584 (D.C. Cir. 1971).

Prior to analysis of benefits, it must be kept in mind that the risk we are dealing with is that of cancer, a matter of grave concern. "Environmental Defense Fund v. Environmental Protection Agency," 465 F. 2d 528, 538 (D.C. Cir. 1972); "Environmental Defense Fund v. Ruckelshaus, supra." Moreover, we must seriously heed the admonition of the Court in the latter case wherein it is stated that the Delaney Amendment to the Federal Food, Drug, and Cosmetic Act indicates "the magnitude of Congressional concern about the hazards created by carcinogenic chemicals, and places a heavy burden on any administrative officer to explain the basis for his decision to permit the continued use of a chemical known to produce cancer in experimental animals." 439 F. 2d 584, 596, fn. 41 (D.C. Cir. 1971).

On the other hand, we must seriously consider the 1975 corn crop, especially in view of the drought this year which has somewhat diminished expectations, its importance and the possible effect of a ban of the use of aldrin thereon during the time it will take to issue the final decision in the consolidated cancellation proceedings. As seen from the Findings of Fact, aldrin use far exceeds that of dieldrin and the major use of aldrin is on corn.

Corn is the world's principal grain used for cattle, hog and poultry feeding and is an important food grain as well in certain countries. We are extremely conscious of the importance of the 1975 corn crop to protein food production and the economy. World grain stocks are at the lowest level in more than 2 decades. Despite generally larger crops elsewhere, the smaller than expected U.S. corn crop this year due to weather conditions will prevent rebuilding world stocks this year. It will be necessary to await next year's crops before there can be hope of rebuilding such stocks.

But, we do not believe that the availability of aldrin or lack thereof will significantly affect the 1975 corn crop. Stated another way, it appears to us from the record that the necessity for aldrin in the production of that crop and the consequence of its unavailability have been exaggerated.

To place our inquiry in proper perspective, it should be noted that aldrin is utilized on only approximately 8–10 percent of the acreage devoted to corn production and that some of its use thereon is actually unnecessary. In other words, aldrin is often applied as "insurance." As with much insurance, the covered risk does not occur and would not have occurred even in the absence of the insurance coverage. This is not to say that in certain situations the need for insecticides is not more apparent than in others. In addition, there is some evidence of record that corn soil insect populations are at low levels.

Dr. John Schnittker, a former Under Secretary of Agriculture of the United States who has much experience and expertise with respect to the economics and marketing of feed grains testified on behalf of respondent in these proceedings. He assumed, for the purpose of his testimony, that the absence of aldrin would result in a 1, 2 or 3 percent diminution in the corn crop and projected the consequences of such reductions.[29]

Dr. Schnittker's testimony indicated that the overall economic effects of the ban of aldrin for use of corn depend to a great degree on the extent of the future demand for grain imports which will be placed on the United States by other countries, as well as on a variety of facts affecting the supply of corn, such as the supply of suitable land, technological developments in corn breeding and husbandry, demand for other agricultural products under soil and climatic conditions to which corn is well adapted, federal farm programs, weather conditions and fertilizer availability. The unpredictability of such factors as weather make projections about future corn harvests in specific years extremely difficult as the recent drought in the corn belt demonstrates. In this connection, however, it appears to us that the reduction of the 1974 corn crop below expectations would, in terms of Dr. Schnittker's analysis, result, in effect, in shifting, in part, his estimates and consequences for 1974 to 1975 since the same basic capability to produce a corn stockpile from next year's crop would remain.

Dr. Schnittker concluded that the current situation prevailing in the grain market is abnormal and short term, resulting from the somewhat unprecedented crop shortfall of world grain in 1972 and 1973 which necessitated a depletion of accumulated reserves. He predicted a general reduction in the import of grain by all countries because "the magnitude of the decline in world grain production in 1972 appears to have been principally the result of events which should not be expected to recur regularly." He further stated that "the analysis of agricultural production potential and targets * * * leads to the conclusion that success in expanding production is possible and probable in most countries and that U.S. grain exporting capacity will not be tested every year until the end of the 1970's."

In short, Dr. Schnittker found very little macroeconomic effect of even a 3 percent reduction in corn production, a reduction which he considered to be well beyond any known estimate of the actual impact to be expected from the unavailability of aldrin. We are in full agreement with both of these conclusions. This is not to say that we are not very concerned about possible effects of suspension upon individual farmers, a matter we shall discuss in the next part of these Conclusions.

While predictions and projections are hazardous for obvious reasons, it appears that the planting of additional acres to compensate for any reduced yields would nullify any price impact at the national level and even if no additional acres were planted to offset any yield impact the price of corn would increase by only 1.5 to 5.8 percent for the 1 and 3 percent reductions.

But, as indicated by footnote 29, we do not believe that a 3 percent reduction in yield could result from the absence of aldrin in 1975. In fact, we seriously doubt that even a one percent decline would result. We have been casting about in these proceedings for a reliable estimate of the reduction in yield that would be attributable to a suspension or cancellation of the use of aldrin in the production of corn. One of the obvious problems in this connection is an inability to determine what would have been the case if aldrin had not been used. Aldrin is in part utilized by farmers as "insurance" and may not have been actually necessary at least in some very substantial number of instances.

We totally reject the Doane Agricultural Service, Inc. special survey and projections of loss adduced by the Shell Chemical Company. On its face, it is patently exaggerated, employs "double counting compounded," is based on a small sample from which amazing projections are made and elicited the views of aldrin users who would not in reality know with any precision the effects of the absence of aldrin and who, it seems to us, would demonstrate a bias. Such survey, it also seems to us, was biased in its design, responses and presentation of the survey questionnaire and results and displayed other weaknesses such as statistical deficiency. Similarly, the very rough study of Dr. Freund, which was only intended to be a tentative and preliminary work, cannot be relied upon as indicated by the report itself which states that "the assumptions are extensively qualified and for firm conclusions, more data on many aspects of the study are needed."

It appears to us that aside from the matters mentioned above, the only economic study offering some reliance is that of Dr. Herman W. Delvo, Agricultural Economist, National Economic Analysis Division, Economic Research Service, United States Department of Agriculture, entitled "Economic Impact of Discontinuing Aldrin Use in Corn Production," issued June 1974. Dr. Delvo uses data accumulated from the USDA 1971 Farm Production Expenditure Survey to establish the use pattern of aldrin in 1971. He relies on consultations with entomologists in the Corn Belt states to estimate overall losses in the event that aldrin, heptachlor and chlordane were not available for use.[30] Dr. Delvo estimates that the overall loss in 1971 in corn production would have been 19 million bushels for the Corn Belt states and a total of 21 million bushels for the entire nation where farmers make use of alternatives to aldrin (But, see footnote 30). If farmers did not use alternatives, he estimates the 1971 loss at 51 million bushels for the Corn Belt states and a total of 55 million bushels for the United States. Even adjusting for 1975 increased acreage over 1971 acreage and considering the fact that, perhaps, use of aldrin for rootworm control may have been overstated, the estimated loss in this study where farmers utilize alternatives is much below one percent of estimated production and may reach one percent if alternatives, including heptachlor and chlordane, are not used, a situation which is improbable.[31]

---

[28] As indicated above, there was no reversal of position with respect to heptachlor and chlordane availability.

[29] Such testimony was received in the cancellation proceedings, and his projections were not specifically related to the 1975 crop. Nevertheless, they are valid for these suspension proceedings. In any event, we cannot conceive of a 3, 2 or perhaps even a 1 percent reduction in the 1975 crop by virtue of the absence of aldrin. There is in reality no good basis in the record to predict such a loss probably approximating over 60, 120 and 180 million bushels of corn at the 1, 2 and 3 percent reduction levels, respectively. (See discussion which follows).

Confirmatory of the general conclusion of Dr. Schnittker, Dr. Delvo found very little in the way of macroeconomic effect resulting from the absence of aldrin as an input in corn production. In fact, he found a 0.8 percent increase in price with use of alternative insecticides and a 2.2 percent increase in price without alternative insecticides with farmers showing a net gain.

On the basis of the foregoing, we cannot find any major economic or social benefit resulting from the use of aldrin on corn in 1975 in the context of overall effect of its unavailability for such use. In other words, we could not meet the burden placed upon us for continued use by the Court in "Environmental Defense Fund v. Ruckelshaus," supra at footnote 41.[32] It would be strange, indeed, to allow the use of aldrin for the 1975 corn crop and thereby continue to jeopardize the health of the American people in order to place a relatively small amount of corn into the world stockpile. Concern expressed for starving people abroad can be met or satisfied by other means it seems to us, if necessary.

VII. We turn now to the impact of the absence of aldrin upon individual corn farmers, also a matter of great concern. It must be remembered in this connection as well that considerable quantities of heptachlor and chlordane will be available in 1975 and those farmers who feel a need for aldrin may avail themselves of these alternates to some extent.[33]

Initially much was said of the "corn soil insect complex" consisting of some 20 soil insects that attack corn. Upon analysis, however, it appears that there are generally only 3 and possibly 4 insects that can be of economic significance with respect to damage to corn, namely, the corn rootworm, cutworm, wireworm and, perhaps, the white grub. These insects have varying degrees of importance. The other soil insects attacking corn are not usually even treated for with pesticides, Shell Chemical Company did not include them in a proposed or suggested limitation on use offered by it in these proceedings and we shall confide our consideration to those insects specified above.

The corn rootworm is by far the major corn soil insect pest in the Corn Belt and attacks continuous, as distinguished from, first year corn. Two of the 3 varieties or species of the corn rootworm, that is, the Western and Northern corn rootworm, are now resistant to aldrin and are found in much or most of the major corn producing area of the country. There are many organophosphate and carbamate insecticides which effectively control the resistant corn rootworm and also the nonresistant variety. Consequently, we do not consider the corn rootworm in our determination with respect to the need for aldrin as this pesticide is not used in much of the Corn Belt for the control of this insect and to the extent that it is so utilized to control the nonresistant corn rootworm it may readily be replaced by those chemicals employed to control the resistant variety.[34]

The next major soil insect pest of corn is the cutworm and we shall discuss it below. The wireworm and, perhaps, the white grub are also economically significant pests of corn but to a much lesser degree than the rootworm or the cutworm. On the basis of the record, we are not overly impressed with the importance of the wireworm. Dr. Delvo, in the study referred to above, found a reduction of only 2,556,000 bushels of corn in the United States due to wireworms if alternatives other than heptachlor and chlordane were used. This seems too high and this figure would need be much less if these 2 insecticides are included in the alternatives. Unlike the cutworm, the wireworm appears to be associated with cropping patterns where corn is grown after sod or pasture. It is a problem primarily of first year corn but can be found in second year corn following sod where it was not properly treated the prior year. Respondent proposes various preplant soil incorporated pesticides as alternates to aldrin for control of the wireworm. Such alternatives are registered for such use[35] and have shown effective results in field tests. Several of these proposed alternatives performed better or more effectively than aldrin in these field tests. In fact, the record demonstrates some question as to the effectiveness or consistency of aldrin in wireworm control. We do believe or agree, however, that there may be questions with respect to the consistency of effectiveness under all conditions of the alternatives, but under the circumstances presented in this proceeding they properly must be considered as viable alternatives.[36]

Additionally, as we have stated, the wireworm is generally only a significant problem to the individual farmer when certain rotations are followed. Since we are only concerned herein with the 1975 corn crop/the farmer, if he anticipates problems in the absence of aldrin and does not care to apply or cannot obtain one of the possible alternatives including heptachlor and chlordane, may to a large extent solve his problem by the rotation he chooses.[37] For example, a farmer may grow soybeans a second year, a crop which is not greatly affected by the wireworm, or may plant sod or pasture in soybeans rather than starting initially in a corn-soybean rotation, although there is probably very little sod or pasture now available. However, the corn-soybean rotation is probably the most insect free and does not present a great wireworm problem in the rotation from soybeans back into corn. We recognize that this may somewhat restrict some relatively few farmers, but, in the context of these proceedings, such restriction is necessary. As much of the corn land is in continuous corn, we do not believe that great numbers of farmers are faced with this choice absent the availability of aldrin in 1975.

The insect which gives us most concern in connection with its affect upon the individual farmer is the cutworm. Unlike the wireworm, the cutworm is generally a problem associated with geography, soil and weather. In other words, the cutworm is associated generally with poorly drained river bottom land, heavy soils and low wet spots in upland fields, and rotation does not play a major role in connection therewith except to some extent on first year corn following sod or legumes.

---

[30] In the consolidated cancellation proceedings, heptachlor and chlordane, which are admittedly as effective as aldrin, were not proposed as alternatives thereto and our considerations therein were limited to alternatives to aldrin other than these 2 insecticides. It appears, however, that approximately 3,000,000 and 1,000,000 pounds of technical heptachlor and chlordane, respectively, will be available for use on corn in 1975. We cannot ignore such fact in assessing the effect of a suspension of aldrin on the 1975 corn crop. Consequently, Dr. Delvo's estimates must be considerably reduced since he did not include heptachlor or chlordane in arriving at his conclusion. In reality, his estimates of loss if farmers used alternatives must be reduced, perhaps by 20 to 40 percent or more as testimony in these proceedings indicate that farmers would switch to heptachlor and perhaps, chlordane, the efficacy of which is not in question. In addition, such estimate was based on the supposition that the other alternatives would not be as effective as aldrin. This may not be so at least with respect to newer alternatives for use against the wireworm. We make further observation that many and, perhaps most farmers do not apply aldrin as directed for heavy wireworm or cutworm infestations and losses from such infestations might not be so different with aldrin or an alternate treatment. Also, we note that some of the entomologists with whom Dr. Delvo conferred have testified in these proceedings for Shell or for respondent.

[31] We do not at this time know corn plantings for 1975 but we assume that they should approximate 1974 plantings and that production estimates should be similar for both years especially in view of the present price of corn. In this connection, we also believe that most farmers will use alternate chemicals, even if more expensive than aldrin, because of the favorable price picture and the fact that pesticides represent a relatively small part of the cost of production. In any event, we cannot make estimates of, or on the basis of, failure of farmers to use alternatives. Most importantly, we do not necessarily agree by virtue of the above analysis that losses would be as high as stated by Dr. Delvo. We have merely used his paper as a frame of reference. We believe that Dr. Delvo may have overestimated losses due to wireworm and cutworm damage.

[32] Shell Chemical Company in its pretrial brief in the consolidated cancellation proceedings did not, in reality, contend for a macroeconomic effect resulting from the absence of aldrin and several of the entomologists called by Shell as witnesses agreed that its unavailability would not have such an effect in their states.

[33] We do not consider in these suspension proceedings, as distinguished from a cancellation proceeding, such basic questions as biological control without the use of insecticides, possible new resistance of insects to aldrin, possible resurgence of insect populations absent aldrin, etc.

[34] Respondent advances a theory that the substitution of organophosphate and carbamate insecticides for aldrin for control of the nonresistant rootworm may result in increased yields. Such position is too speculative for adoption.

[35] We cannot, it seems to us, consider promising alternatives that are, perhaps, in the "registration pipeline" but are not as yet registered. Mention should be made, however, of section 3(f)(2) of the act (7 U.S.C. 136a(f)(2)) which provides, in part, that "as long as no cancellation proceedings are in effect registration of a pesticide shall be prima facie evidence that the pesticide, its labeling and packaging comply with the registration provisions of the Act." We should state, however, that promising additional alternatives are in the "pipeline" and we surmise that they probably will be registered for the 1975 season.

[36] Alternatives, other than heptachlor and chlordane, are listed in the Findings of Fact. We do not rate their respective merits. The farmer concerned about wireworm damage must consult his state extension entomologist for recommendations with respect to his in-

[37] Rotation can also solve the problem of the billbug and white grub to a great degree.

There does not appear to now be an effective preplant or planting time insecticide for the control of the black cutworm, although several insecticides with unknown effectiveness are in the "registration pipeline." Instead, the currently available alternative to aldrin preplant treatment under corn is the application of post emergent sprays and baits, that is, after the insect has actually appeared. Philosophically and as a practical matter, this method of treatment has the advantage of treating for known insect infestation and the avoidance of an "insurance" treatment of entire fields where the insect may not appear, may only attack part of a field or may appear but not in numbers of economic significance.

An entomologist presented by the Shell Chemical Company and others testified that the post emergent baits are as effective as, or better, than, a one pound per acre hand or row application of aldrin against the cutworm which is not as effective as a 2 pound per acre broadcast application thereof. The lower rate of application of aldrin is not effective against a heavy black cutworm infestation, but many, if not most, of the farmers apply aldrin at the lower rate. In other words, they are willing to settle for less than the best treatment. This should be a factor, perhaps, in evaluating the sprays and baits as substitutes for aldrin and the actual necessity for any treatment. In any event, the record supports the conclusion that post emergent treatment of black cutworm, the major cutworm pest, with baits is efficacious with post emergent sprays having lesser effectiveness.[illegible]

However, post emergent treatments for the black cutworm has several difficulties or disadvantages. In order to be effective as alternatives to preventive preplant or planting time applications of aldrin, the baits or sprays must be timely applied. This requires that the farmer observe his fields carefully during an approximate 3 to 4 week period when the corn begins to emerge. This does not mean that all farmers need observe their fields or that those farmers with a suspect cutworm problem need observe all their fields. It does mean that the farmer who has had cutworm problems in the recent past must check key survey spots in his suspect fields. While a 6-state cooperative survey is developing a scouting system, we are not, we believe, at the point of having available commercial scouts or commercial scouting of farmers fields for cutworms. Rather, the individual farmer, his family or employees or even high school students could scout or walk select portions of corn fields in an attempt to detect early signs of cutworm damage. Such damage is more readily recognizable than damage caused by other insects. We recognize that this imposes a burden on the farmer at perhaps his busy time of year.

Concomitant with early detection of cutworm infestation is the necessity for rapid treatment with baits or sprays. If the farmer observes early cutworm feeding damage he has several days in which to apply a bait or spray insecticide to protect the crop. The baits will then prevent further loss of stand and any cut corn will have an opportunity to regrow. However, under extreme dry or wet conditions, the bait insecticides may lose some of their effectiveness. An entomologist presented by respondent testified that 75 to 80 percent of the Illinois corn farmers have obtained good to excellent black cutworm control with post emergent baits.[illegible]

As can be seen from the prior discussion, the use of post emergent baits and sprays in lieu of aldrin presents extra effort and some additional uncertainty. We do not want to leave the impression, however, that cutworm loss is irreparable. Should a field or a portion of a filed suffer serious cutworm damage, the farmer has the option of replanting corn thereon. In fact, this is usually done. It is recognized that in that event the farmer suffers the costs of replanting and suffers some loss of yield due to the later planting. But, with the current price of corn, the farmer will most likely receive a profitable return from his corn production, which return will, of course, be reduced from what he would have experienced. In fact, a farmer may initially plant corn later on suspect acres and, perhaps, avoid cutworm injury. In this event, he would suffer some loss of yield due to late or later planting. In addition, heavier seeding is also a valid measure the farmer can take.

Farmers generally are not that familiar with the use of post emergent treatments or with scouting. There appears to us adequate time to prepare for such matters prior to planting time in 1975 which shall probably begin around April 15, 1975.

We do not lightly make these findings as we do not desire to cause additional burdens and uncertainty to farmers who have a history of cutworm problems.[illegible] But, it appears to us that there is a relatively adequate alternative to aldrin in the treatment of the black cutworm and therefore we cannot conclude that during 1975 aldrin use should be continued for this purpose in view of our conclusions as to the risks accompanying aldrin.[illegible] We do not expect the corn farmer who has cutworm problems to like this conclusion. We have, perhaps, imposed some onerous burdens upon him. The act makes this requirement, we believe. If the post emergent alternative is not acceptable to some farmers of bottom land, they have the option, perhaps, of planting other feed crops during that season, including soybeans which is another important feed crop. Should some of this acreage be lost to corn in 1975, the replacement thereof by some other feed crop is merely a trade off we believe.

To summarize, we cannot justify the use of aldrin under corn in 1975 both from a macroeconomic or microeconomic standpoint.

VIII. Aldrin is also utilized for control of the Fuller Rose Beetle in Florida, one of the more minor citrus pests in that State. While sales statistics adduced by the Shell Chemical Company indicate that this insecticide is sold and used on citrus in much of Florida, expert witnesses presented by Florida Citrus Mutual, a major grower organization, testified that the economic significance of the Fuller Rose Beetle is very circumscribed geographically in that State. Of the 877,000 acres of citrus in Florida, the rose beetle is only present in numbers sufficient to commence to reduce yield on between 10,000 and 50,000 acres. The area of significant infestation is essentially the Indian River area of the Southeastern seaboard of Florida, an area characterized by poor internal soil drainage, a high water table, and consequently unusually shallow citrus root systems. Less than 5 percent of the total citrus acreage in Florida has been treated with any soil insecticide for control of any insect and even within the Indian River Fuller Rose Beetle trouble region only 20 percent of the acreage has been so treated.

In a typical Indian River grove, approximately 75 percent of the feeder roots of citrus trees are located within 18 inches of the top of the ridge of soil upon which citrus trees are usually planted in that area. Such trees are distinguishable by particularly restricted root systems with unusually limited supplies of feeder roots. These systems are less able to make do with decreases in root productivity resulting from insect damage which would be insignificant in other regions within the State of Florida.

Aldrin is overused on citrus to some extent in that it is unnecessarily utilized. Substantial reduction in crop yields caused by lack of treatment for the Fuller Rose Beetle is relatively rare when the industry is considered as a whole.

As indicated in the Findings of Fact, cultural practices offer a large potential for disruption of pest problems caused by the rose beetle and alternative insecticidal foliar sprays, most of which are already used in the Florida citrus program, some as often as 4 to 6 times a year, provide good initial kill of the adult weevil. The State of California does not recommend the use of aldrin to control the rose beetle on its very substantial citrus acreage and a large Florida citrus grower organization does not utilize it.

Once again, we need put the issue with respect to the continued use of aldrin or dieldrin on Florida citrus in perspective. We are presented herein in these suspension proceedings with the limited question of its continued use during the time it would take to complete proceedings relating to cancellation of such chemicals. We are talking, it seems to us, of one split application of aldrin or at most one annual application thereof.

It is clear from the record that in view of the limited area of possible need and, in reality, the limited number of orchards or trees involved, the absence of aldrin during the restricted period of consideration would

---

[illegible] Most states now recommend the post emergent treatment as an emergency treatment. It should be stated at this point that Wisconsin has banned the use of aldrin on corn and that the Illinois state recommendations do not inculde aldrin. Instead, the post emergent treatment is recommended.

[illegible] The sandhill and glassy cutworm cause special concern as they are subterranean feeders and the bait is probably inadequate. These cutworms are not widespread and there is some indication in the record that band treatment of Dursban, Dyfonate, Mocap and Diazinon could be effective as to them.

[illegible] Another uncertainty presented for the record is the availability of the alternate insecticides in 1975. There need not be a pound for pound displacement especially with respect to post emergent treatment. Heptachlor and chlordane are available, insect populations appear to be at low levels, rootworm insecticides give some control of wireworms and perhaps cutworms and aldrin has been overused in the past. We agree that the situation will be tight. We also believe, however, that this decision will generate some additional alternate pesticides to the extent that is possible. Also, any existing stocks of aldrin, if any, could be utilized and the intermediates contracted for by Shell could possibly be available for additional heptachlor production.

[illegible] Some of the parties primarily in the cancellation proceedings have taken the view that proposed alternatives need be as efficacious as, and no more costly than, the chemical at issue. We reject such a standard especially when the risk at hand is as ominous as cancer.

have little, if any, affect upon the Florida citrus industry or the price for its products. Additionally, we see very little effect upon the relatively small number of possibly affected growers. Cultural practices and foliar sprays are available to them as alternatives to aldrin or dieldrin.[42] Further, we surmise that existing stocks of these products, the use thereof not being barred by the Administrator's August 2, 1974 notice of suspension, may well be present in Florida to some extent. In short, we see no overriding benefit or any great disruption from the nonavailability of aldrin or dieldrin for Florida citrus during the next growing season.[43]

IX. Aldrin and predominantly dieldrin are also used for seed treatment or dressing on many different types of seed. The record is not as complete with respect to the need for these insecticides in the treatment of some seeds as distinguished from others or with respect to seed treatment generally. Certain generalizations can be made however. Farmers will purchase seed after it has been treated commercially, will treat the seed themselves prior to planting, often as part of a slurry or liquid mixture, or will add the chemical directly to the seed in a planter box at the time of planting. Commercial treatment of seed is more practical, tending to provide a more even and effective distribution of relatively small quantities of insecticide, particularly in contrast to individual grower's applications by means of planter boxes.

Dosages vary according to the type of seed treated and the seeding rate per acre. Under normal conditions or circumstances, aldrin/dieldrin is applied to seeds at the rate of one-half ounce to one ounce of the chemical per bushel or per 100 pounds of seed. The cost of seed treatment with these insecticides is relatively small and, in some instances, is not passed on to the farmer.

Dieldrin has an effective life as a seed dressing in soil of approximately 10 to 20 days. In warm or hot weather, seeds will typically germinate in 4 to 5 days, but in cool, damp weather germination may be delayed to a week or 10 days. Most of the seed dressing alternatives advanced by respondent are less persistent than dieldrin and provide less of a margin of protection. Lindane appears to be an effective alternative but for some criticism of a delay in germination of the seed resulting from its use. This apparently occurs if the seed has been treated with lindane sometime, such as 3 weeks, before planting. A simple answer to this criticism is a planter box application of lindane by the farmer at the time of planting. This process, of course, has some of the disadvantages mentioned above.

Here too, however, we find no compelling macroeconomic or microeconomic reason necessitating the use of aldrin or dieldrin seed treatment during the period it will take to complete the consolidated cancellation proceedings. Several viable alternatives are available.

X. In the consolidated cancellation proceedings, the United States Department of Agriculture defended the continued use of aldrin and dieldrin for certain uses in addition to those discussed above and it similarly does so here. These include such uses of one or the other of these insecticides as on Puerto Rican pineapples, sugarcane and bananas, onions grown in the Tulelake Basin of Northern California, strawberries in Oregon and Washington, the Department's quarantine program, cranberries and nursery use.

The parties, that is, in this connection, USDA, respondent, Environmental Defense Fund, Inc. and the National Audubon Society, are, in effect, attemtping to place us in the straitjacket of deciding the ultimate issues presented by the uses involved in the consolidated cancellation proceedings. We refuse to be so restricted. For this reason the briefs filed by these parties do not, in great measure, really address the problem at hand.

We have stated several times in this Decision that we are solely presented with the continued use of aldrin/dieldrin during a relatively limited time frame, the time it will take to complete the cancellation proceedings. We do not intend to consider matters beyond that period in this Decision. In addition, the briefs of these parties with respect to these uses do not deal with the significance of the availability of heptachlor and chlordane in 1975. For the most part, the parties attack the issues as if heptachlor and chlordane do not exist. This is absurd and we have no intention of deciding the questions posed herein as if they do not exist because the "real world" situation cannot be ignored.

Heptachlor and chlordane were not proposed as alternatives by respondent and the Environmental Defense Fund for the reasons explained earlier. But, these chemicals are here and are registered for many of the uses defended by USDA. In reality, USDA does not challenge or question the efficacy of these insecticides for most of their registered uses. In fact, it recommends the use of chlordane in its regulatory and control programs and "dieldrin is reserved for those limited uses involving soil surface treatments * * * where chlordane will not render the required 100 percent control. * * * This reflects Departmental policy requiring that chlordane be substituted for dieldrin wherever possible."[44]

We need not analyze each of the USDA defended uses and the need for aldrin or dieldrin thereon. Heptachlor or chlordane are registered and effective for such crops or uses as pineapples, greenhouse, nurseries and nursery turf, onions, perhaps strawberries, sugarcane and apparently bananas. Additional substitutes are also available for some of these and other uses. Also, there are alternatives in the "registration pipeline" which we surmise will receive priority.

It can also be stated with respect to the uses involved that we see no major food supply problem and certainly no macroeconomic effect from the lack of aldrin or dieldrin. In fact, the cranberry industry is currently suffering from a glut or oversupply. Also, we see no substantial microeconomic consequence from the absence of these pesticides during the limited period at issue. Actually, the absence of any insecticides in some instances will not have effect for some years. It must be realized in this connection that aldrin or dieldrin are not used annually with respect to most of these crops and the affected growers represent a small segment of those industries. For example, a minimum of 5 year protection is claimed with respect to cranberries. In short, we believe that the growers involved can manage for one season at most without aldrin or dieldrin but with the alternatives at hand. As to some of these growers, a different crop rotation is available if they are convinced that they cannot do without aldrin or dieldrin and cultural practices are available to negate or minimize the absence thereof. For example, flooding of cranberry bogs can eliminate the insect pest or pests.[45] In addition, to the extent existing stocks of aldrin and dieldrin are available, they may be used.

To summarize, there clearly does not exist any compelling reason to make aldrin or dieldrin available in 1975 for the uses defended by USDA. We are not hereby saying that our conclusions with respect thereto will be the same in the consolidated cancellation proceedings when we assume that heptachlor and, perhaps, chlordane will once again not be considered as alternatives. We can foresee, for example, a possible conclusion calling for continued use of aldrin or dieldrin at least for a limited period of time while alternatives are found. The record demonstrates in most instances inaction or inadequate action in this regard.

In addition to all of the uses of aldrin and dieldrin already discussed in these Conclusions, they are uses for which no evidence has been adduced with respect to the benefits to be derived from, or the need for, continued use of these insecticides. It is patent, therefore, that there exists no basis to judge such benefits and that, in the context of these proceedings, no economic, social or environmental benefit results from the continued use of these pesticides for such purposes.

XI. Shell Chemical Company, in its objections, alleges certain procedural defects or irregularities in the issuance of the Notice of Intention to Suspend by the Administrator August 2, 1974, which set in motion the institution of these consolidated suspension proceedings. First, it contends that such notice reversed 2 previous decisions by a former Administrator that aldrin/dieldrin was not an "imminent hazard" allegedly on the basis of the same evidence before the present Administrator. USDA similarly makes this argument.

In his Determination and Order of December 7, 1972, in the consolidated aldrin/dieldrin cancellation proceedings the prior Administrator, in deciding not to suspend such insecticides stated, in part, that "the present evidence, confined to one strain of mouse is tentative evidence of a 'risk,' but not sufficient proof that aldrin/dieldrin is a carcinogen in human beings. If unrebutted, this evidence would be a caution signal as to long-term exposure, but does not amount to a red light requiring immediate elimination of all dieldrin residue in the diet." The Administrator in his August 2, 1974, Notice of Intention to Suspend stated that "an intense examination of the relevant evidence over the past year * * * brought to light certain previously unknown facts, which have now been reviewed and scientifically documented for the first time." Such facts, clearly additional to those mentioned by the former Administrator in his order of December 7, 1972 and his order of March 18, 1971, in which he also failed to suspend the 2 insecticides involved, are then briefly set forth in the August 2, 1974 notice of the Administrator. They clearly form a new and additional basis supporting and, perhaps, requiring the notice of intention to suspend. Further, certain factual assumptions or predictions by the former Administrator forming the basis for his decisions not to suspend proved to be untrue. Moreover, the Administrator could also issue such a suspension on the basis of an extensive re-evaluation of existing information "which perhaps brought its full impact to the attention of the experts for the first time." "Bell v. Goddard, supra," at p. 181.

---

[42] We have some hesitation or reservation with respect to some possible disruption of an integrated pest management control system employed in Florida in the control of other insects by the use of alternative foliar sprays. We are not aware that this would necessarily occur however.

[43] Two other weevils of lesser economic consequence than the rose beetle were mentioned in the record. All we have said with respect to the Fuller Rose Beetle is applicable thereto. In addition the diaprepes abbreviatus eradication program has available to it several alternatives.

[44] Chlordane surface application is admittedly effective for nurserymen where certification is unnecessary. The alleged need for dieldrin surface application in limited circumstances for certification status for a 4 year period can surely be solved by USDA during the limited period involved herein if only by an additional application of chlordane. This circumstance should not arise often during the limited period and we are certain that administrative adaptability and ingenuity will easily solve this temporary problem.

[45] There are no registered alternative chemicals for use on cranberries. But, very few, if any, growers should critically need the chemical in 1975. Only 300 acres were treated in Massachusetts in 1972.

Shell Chemical Company further contends that the Notice of Intention to Suspend "is fatally defective in that, on information and belief, it was based on improper *ex parte* communications with the Office of the Administrator by parties in the cancellation proceeding and/or their representatives or agents and/or Congressmen and Senators and their staffs."[66] These allegations have not been established. In any event, they are bottomed upon Shell's contention that the suspension proceedings are but a phase or part of the cancellation proceedings. Such is not the case. We agreed that the August 2, 1974 notice was based in large part upon evidence adduced in the cancellation hearing. This does not alter our conclusions. It would be nonsensical to suggest that the Administrator could not consider such evidence in making his determination to suspend or that he need hold, in effect, a public hearing on question of whether a suspension proceeding should be instituted which would in turn require a public hearing, which Shell appears to contend herein.

The Administrator, in the issuance of the August 2, 1974 notice, was functioning in an accusatory capacity in instituting or initiating an action with the further responsibility of ultimately determining the merits of the "charges" so presented. While what was formerly known as the Administrative Procedure Act requires the separation of the adjudicatory and prosecutorial functions in an agency (5 U.S.C. 554(d)), it does not prohibit the combination thereof in the determination as to whether a proceeding should be instituted. See e.g., "Federal Trade Commission v. Cinderella Career and Finishing Schools, Inc.," 404 F. 2d 1308, 1315 (D.C. Cir. 1968) and cases cited therein; "Amos Treat & Co. v. Securities and Exchange Commission," 306 F. 2d 260, 266 (D.C. Cir. 1962); "R. A. Holman & Co. v. Securities and Exchange Commission," 366 F. 2d 446, 455 (2d Cir. 1966). It may well be that the Administrator should not ultimately decide the aldrin/dieldrin consolidated cancellation proceedings if *ex parte* contact was held with those engaged in investigative or prosecuting functions in those proceedings in determining whether a suspension proceeding should be instituted.[67] But, we see no impediment by reason thereof in his acting in his quasi-judicial capacity in these suspension proceedings.

The United States Department of Agriculture sees additional procedural defects. In its brief, it states, in part, that the consolidated cancellation proceedings would have been finally resolved antecedent to any further significant use of aldrin or dieldrin, that the proponents for cancellation and now suspension had one year to present their case while those defending the continued use thereof only had a very short period of time evidencing a lack of due process, and that these proceedings are defective because the hearing herein began on Wednesday, August 14, 1974 instead of Monday, August 12, 1974, requiring apparently a dismissal thereof. We find little merit in any of these contentions.

As the Administrative Law Judge presiding at the consolidated cancellation proceedings, we had serious doubt as to whether the cancellation proceedings could be completed prior to April 15, 1975, the time of the beginning of corn planting, in view of the time provided in the rules of practice for post hearing procedure, the many additional witnesses to be presented by Shell Chemical Company, the rebuttal evidence that would undoubtedly be adduced to say nothing of surrebuttal, and the extensive cross-examination afforded the parties in those proceedings. These feelings or fears were expressed in ruling on Shell's motion in this connection at the hearing herein. Even if completion were possible by then, which is doubtful, some 6 to 10 million pounds of technical aldrin[68] or approximately 30 to 50 million pounds of the formulated product would have had to be disposed of if the Administrator concluded that aldrin registrations should be cancelled.

USDA's contention that the proponents for cancellation and now suspension had a year to present their case is a glaring over statement and distortion. The case of respondent and EDF on human health took perhaps a little over a month and that was due in great measure to extensive cross-examination conducted by the Shell Chemical Company. The environmental case was not incorporated into the suspension proceedings. In addition, Shell Chemical Company and USDA incorporated by reference much evidence from the cancellation proceedings into the suspension proceedings and did not thereby lose the benefits of their presentation in the cancellation proceedings. It is true that the Shell Chemical Company put on its case with respect to cancer in a shorter period of time than respondent and EDF, but that was due in great part to the fact that while Shell could extensively cross-examine in the cancellation proceedings, the cross-examination by respondent and EDF was greatly restricted by time contraints in the suspension proceedings. It seems to us, as we stated at the hearing, that Shell received some benefit or advantage as the result of those circumstances.

Finally, USDA contends that Shell Chemical Company was entitled by the statute to have the suspension hearing begin on August 12 instead of 14, 1974, when it did begin, and that a dismissal of these proceedings is warranted thereby. We agree that pursuant to the act Shell Chemical Company was entitled, perhaps, to have the hearing begin on the earlier date. As we explained at the prehearing conference herein, the act was drafted on the basis of a single objector to a notice of suspension. As we further stated, we were concerned with the rights of the over 20 additional objectors to the notice of suspension who are located outside of Washington, D.C. and who received notices later than the Shell Chemical Company. We do not believe that a 2 day delay under the circumstances presented, as spelled out in the transcript of the prehearing conference, is in error, prejudicial to Shell Chemical Company or of substance. To begin the hearing on August 12, 1974 could well have been prejudicial to the many other objectors, some of whom did not even have to file objections until August 12, 1974 or later.

XII. The ultimate question is now presented, that is, whether the continued use of aldrin/dieldrin during the time it will take to complete the consolidated cancellation proceedings presents an imminent hazard, that is, "would be likely to result in unreasonable adverse effects on the environment." We are to determine whether an unreasonable risk to man or the environment is likely during the interim period taking into account the economic, social, and environmental costs and benefits of aldrin/dieldrin.[69] Our answer to such query is apparent from all that went before in this Decision. Some of the pronouncements of the Administrator with respect to suspension, in the context of these proceedings, also demand a finding of imminent hazard. In the Reasons Underlying the Registration Decision Concerning Products Containing DDT, 2,4,5-T, Aldrin and Dieldrin, issued March 18, 1971, he stated, in part, with respect to suspension, as follows:[70]

> * * * this Agency will find that an imminent hazard to the public exists when the evidence is sufficient to show that continued registration of an economic poison poses a significant threat of danger to health, or

---

[66] We note in this connection that section 21(b) of the act (7 U.S.C. 136s(b)) provides as follows: (b) In addition to any other authority relating to public hearings and solicitation of views, in connection with the suspension or cancellation of a pesticide registration or any other actions authorized under this Act, the Administrator may, at his discretion, solicit the views of all interested persons, either orally or in writing, and seek such advice from scientists, farmers, farm organizations, and other qualified persons as he deems proper.

[67] We do not hereby necessarily agree with counsel for Shell Chemical Company that section 164.7 of the rules of practice applies to such alleged *ex parte* communications even in the cancellation proceedings.

[68] A representative of the Shell Chemical Company stated at the hearing that it intended to produce 6 million pounds of technical aldrin for use on corn in 1975. We have serious doubt as to this view of the end use estimates of aldrin on corn in 1972, 1973 and 1974.

[69] Our prior discussion did not consider the economic costs the continued use of aldrin and dieldrin pose to the user thereof and others. In short, what we have reference to is dieldrin residues in food and feed at FDA actionable limits or above tolerance levels. A witness from the Food and Drug Administration described significant seizures by the FDA by reason of dieldrin residue levels in food and feed. Dieldrin use is indeed economically costly to portions of the food industry. See also United States v. Ewig Bros. Co., Inc., No. 73-1008 (7th Cir. August 28, 1974). Some of those residues, including residues found in poultry in the recent catastrophic "Mississippi poultry seizure" incident, apparently resulted from misuse, accident or mistake. Cf. Sterns Electric Paste Co. v. Environmental Protection Agency, 461 F.2d 293 (7th Cir. 1972). But, the amount of misuse, etc., may well soon reach or has reached the level of "widespread and commonly recognized practice." See section 6(b) of the act. We need not decide this issue at this time.

[70] See also Suspension of Registration for Certain Products Containing Sodium Fluoroacetate (1080), Strychnine and Sodium Cyanide, issued March 9, 1972.

otherwise creates a hazardous situation to the public, that should be corrected immediately to prevent serious injury, and which cannot be permitted to continue during the pendency of administrative proceedings. An "imminent hazard" may be declared at any point in a chain of events which may ultimately result in harm to the public. It is not necessary that the final anticipated injury actually have occurred prior to a determination that an "imminent hazard" exists.

We need not spin any sophisticated, intricate rationals or argument in this connection, as was done by respondent so well in the brief filed herein, with which we basically agree. In short, suspension is to be based upon potential or likely injury and need not be based upon demonstrable injury or certainty of future public harm. Cf. "Environmental Defense Fund v. Environmental Protection Agency," 465 F. 2d 528, 540 (D.C. Cir. 1972).

Briefly, we are talking of a cancer hazard to man. We must remember, in this regard, the characteristics of a chemical carcinogen such as aldrin/dieldrin, that is, the scientific inability to determine a safe or threshold level for man, the fact that the chemicals are carcinogenic at the lowest doses tested, that residues of dieldrin in laboratory species which developed cancer from dieldrin approximate those residues in the American population, the irreversibility of the carcinogenic effect once set in motion by the chemical carcinogen and the long latency period during which the disease has actually set in and is developing but is not yet manifest Given these characteristics, the risk of injury or harm from the use of the pesticides is present during the pendency of the cancellation proceedings even though the effects of such injury may not be manifested for many years to come. This is precisely what the Administrator had in mind in his March 18, 1971 policy statement set forth above, we believe. In short, the continued use of aldrin and dieldrin even during the limited period with which we are concerned presents a significant potential of an unreasonable risk of cancer in the American public.

In this regard, Dr. Saffiotti said the following: It is likely that Dieldrin residues will contaminate a large proportion of the food supply of the American people for many years to come because of past usage of this persistent pesticide. I am clearly not advocating that a large proportion of the food supply to the American people be eliminated because of its presently unavoidable contamination with Dieldrin. At the same time, as a scientist, I am unable to conclude that the continuing contamination of the environment and our food supply with Dieldrin will not produce in some of us the development of cancers, as it has indeed been repeatedly shown to do so in other mammalians.

We fear that we have exhausted the reader by this time and we know we have exhausted ourselves in issuing this decision within the impossible time constraints imposed by the statute and the rules of practice. We merely further say that the registrations of aldrin and dieldrin properly involved herein should be suspended in order "to prevent an imminent hazard during the time required for cancellation" when "taking into account the economic, social, and environmental costs and benefits of the use of" these pesticides by reason of all that has been already said in this Decision. To hold otherwise is to demand a state of knowledge with respect to cancer which we do not possess.

Nor does the recent decision in "Reserve Mining v. United States," No. 74-1291 (8th Cir. June 4, 1974) alter this conclusion as it is distinguishable from the case at hand. While there are several grounds of distinction, such as the relative absence of asbestos in the population of Duluth, Minnesota, as compared with the almost universal presence of dieldrin in humans at significant levels, and the possible difference between an "unreasonable risk to man" and "demonstrable health hazard," the major distinction, we believe, which was recognized by the Court in Reserve Mining, is the question of burden of proof. In that case, the Court stated that "Plaintiffs have failed to prove that a demonstrable health hazard exists. This failure, we hasten to add, is not reflective of any weakness which it is within their power to cure, but rather, given the current state of medical and scientific knowledge, Plaintiffs' case is based only on medical hypothesis and is simply beyond proof." The Court there was not dealing with a substance intended to be utilized as a poison. Under the Federal Insecticide, Fungicide, and Rodenticide Act, as amended, the Congress, on the contrary, properly placed the continuous burden of proof of safety on the registrant.[23]

Order. The registrations issued under the Federal Insecticide, Fungicide, and Rodenticide Act, as amended, of the pesticides aldrin and dieldrin involved in these consolidated suspension proceedings are hereby suspended.[24]

HERBERT L. PERLMAN,
Chief Administrative Law Judge.

SEPTEMBER 20, 1974.

[F.I.F.R.A. Dockets Nos. 145 etc.]

SHELL CHEMICAL COMPANY, ET AL.

OPINION OF THE ADMINISTRATOR, ENVIRONMENTAL PROTECTION AGENCY, ON THE SUSPENSION OF ALDRIN-DIELDRIN

On August 2, 1974, the Environmental Protection Agency (EPA) issued a notice of intent to suspend the registrations and prohibit the production for use of all pesticide products containing Aldrin or Dieldrin, compounds manufactured exclusively by the Shell Chemical Company (Shell). This notice, pursuant to section 6(c) of the Federal Insecticide, Fungicide and Rodenticide Act (FIFRA),[1] resulted in several weeks of expedited hearings before Chief Administrative Law Judge Herbert L. Perlman, the presiding judge at the on-going Aldrin-Dieldrin cancellation hearing which began in August of 1973.[3] On September 23, 1974, he transmitted to me his recommended decision, including findings of fact and conclusions, which is attached to this decision.

I. BACKGROUND

A. Characteristics and Uses of Aldrin-Dieldrin.[4] Aldrin is the common name of a chemical compound containing not less than 95 percent of 1,8,9,10,11,11-hexachloro-2,3-7, 6-endo - 2,7,8 - exo - tetracyclo $[6.2.1.1^{3,6}.0^{2,7}]$ dodec-4,9-diene. It has been used as a contact and stomach insecticide on a wide variety of crops in diverse locations and situations since its introduction in the United States in 1948. As a pure compound, it is an odorless, white, crystalline solid; technical compounds can be various shades of brown. It is lipophilic, meaning that it has an affinity for fatty body tissue, and is fat soluble. It degrades or metabolizes into Dieldrin.

Dieldrin, a closely related manufactured product as well as a metabolic degradation product of Aldrin, is the common name for a material containing not less than 85 percent of 1,8,9,10,11,11-hexachloro-4,5-exo - epoxy-2,3-7,6-endo-2,1-7,8-exo-tetracyclo $[6.2.1.1.^{3,6}.0^{2,7}]$ dodec-9-ene. The pure compound is also an odorless, white, crystalline solid with a somewhat heavier molecular weight than Aldrin. It also is persistent, is more stable and toxic than Aldrin, and is lipophilic.

Aldrin and Dieldrin both are acutely toxic to humans. Poisoning may occur by ingestion, inhalation, or skin absorption, and serious symptoms may result from the ingestion of as little as one gram (1/28 of an ounce). Symptoms of acute exposures include renal damage, ataxia, tremors, convulsions followed by central nervous system depression, respiratory failure and death. Chronic exposures may result in damage to the liver and other body organs.

During the earlier years of its use in the United States, Aldrin was almost entirely limited to applications on cotton, but in the mid-1950's it was replaced by Dieldrin. By 1963, cotton constituted less than one percent of total use of Aldrin. As of 1971, soil applications for corn accounted for 80 percent of the total Aldrin usage. Other uses included termite control (14 percent), rice seed treatment (3 percent), citrus oil use (1 percent), and miscellaneous applications (2 percent). Production of Aldrin in the first six months of 1974 was 9.7 million pounds, compared to approximately 8.7 million pounds produced for the same period in 1973.

Dieldrin, because it is more persistent, replaced Aldrin on cotton until the boll weevil became resistant to both these chlorinated insecticides in the late 1950's and early 1960's. Dieldrin also was used on house flies and mosquitos, until they too became resistant, and on a variety of other insect pests. The use of Dieldrin has declined from a maximum of about 3.6 million pounds in 1956 to approximately 0.6 million pounds today. The most recent accurate figures for Dieldrin indicate that in 1971 approximately 44 percent was used for termites, 20 percent on fruit foliage, 14 percent for seed treatment, 13 percent on vegetables, and 9 percent for miscellaneous uses, including tobacco and sweet potatoes.

---

[23] We do not agree that this burden was not continued in the 1972 amendments to the act or is altered in a suspension proceeding, as contended by Shell Chemical Company. Mention should also be made of United States v. Ewing Bros. Co., Inc., No. 73-1008 (7th Cir. August 28, 1974) where the Court found that DDT and dieldrin found in processed fish at levels above FDA actionable limits were "food additives" under the Federal Food, Drug and Cosmetic Act. We are uncertain of the significance of this case to the issue at hand.

[24] In order to avoid any ambiguity we have not made any distinction with respect to registrations of aldrin and dieldrin held by registrants in these proceedings which we believe may have already been suspended by operation of law, that is, resulting from the untimely filing of objections. (See footnote 3.)

[1] The Federal Insecticide, Fungicide and Rodenticide Act (FIFRA), 7 U.S.C. 135 et seq., as amended by Pub. L. 92-516, 86 Stat. 973, October 21, 1972. The regulatory authority under FIFRA was transferred from the Department of Agriculture to EPA by Reorganization Order No. 3, 1970.

[3] The transcript of the cancellation hearing already exceeds 24,000 pages, not including many thousands of pages of the witnesses' statements (which are reported separately) and exhibits. The suspension hearing transcript approaches 4,000 pages in length, also not including the lengthy statements by the witnesses and exhibits, which roughly are the same length as the transcripts, plus more than one thousand pages of briefs by the parties.

[4] These two similar compounds have somewhat different uses; but because in the environment or in the body Aldrin quickly degrades to the more stable Dieldrin form, the two terms will generally be used interchangeably in this opinion.

Combined Aldrin and Dieldrin consumption, which in 1970 was 10.7 million pounds, rose in 1971 to 12.3 million pounds. The estimate for 1973 is approximately 11 million pounds.

B. *Definition of cancellation and suspension.* As will be discussed more fully later, cancellation is warranted under the FIFRA when there is a "substantial question of safety" concerning a pesticide. During the period of the administrative review process, which often lasts several years, the manufacture and distribution of the product continues unaffected—a fact which may contribute to the protracted nature of many cancellation proceedings.[4]

Suspension is mandated when there is an "imminent hazard" to man or the environment. This may be declared at any stage of the administrative review process, either upon receipt of new evidence or after reevaluation of existing evidence.[5] The suspension order, which resembles a preliminary injunction,[6] immediately halts the production and distribution of the pesticide and remains in effect until the cancellation hearing is completed and a final decision is made by the Administrator of EPA.

C. *History of the case.* For almost four years, EPA has had under consideration the issue of Aldrin-Dieldrin. On December 3, 1970, one day after the Agency formally came into existence, it received a petition from the Environmental Defense Fund (EDF) requesting the cancellation and immediate suspension of all uses of Aldrin-Dieldrin. As a result, on March 18, 1971, the Administrator of EPA issued a notice of cancellation based upon a finding of a "substantial question as to the *safety*" of Aldrin and Dieldrin.[7] The Administrator also concluded, however, that the evidence then available to him did not demonstrate "an imminent hazard to the public". He, therefore, declined to order a suspension of the compounds pending completion of administrative review.

EDF promptly filed a petition in the United States Court of Appeals for the District of Columbia to review the Administrator's failure to suspend the registrations. The Court's decision, issued on May 5, 1972,[8] remanded the record to EPA for further consideration of the issue of suspension, in light of the judicial interpretation of the power of suspension enunciated in the decision and the March 28, 1972 report of the Aldrin-Dieldrin Scientific Advisory Committee. The Court specifically directed EPA to examine the nature and extent of evidence available on the carcinogenicity of Aldrin-Dieldrin.

Following a review of the scientific evidence requested by the Court, the Administrator reaffirmed the notices of cancellation of nearly all Aldrin-Dieldrin uses on June 26, 1972.[9] The order also solicited public views as to whether any of the cancelled uses also should be suspended, with particular reference to those methods of application and formulation presenting the most obvious risk of widespread, unavoidable dissemination of the compounds.

Five months later, on December 7, 1972, the Administrator announced that the registrants of Aldrin-Dieldrin had agreed voluntarily to eliminate several of the more controversial uses of the product. Furthermore, pursuant to the May 5, 1972 Court of Appeals order, the Administrator announced that he had further examined the issue of suspension and determined that the available evidence still did not justify a finding of imminent hazard.

The cancellation hearing on the risks and benefits of Aldrin-Dieldrin began on August 7, 1973 and was still in progress a year later when, on August 2, 1974, the Agency issued its notice of intention to suspend. On August 7, 1974, a presiding officer, Chief Administrative Law Judge Herbert L. Perlman, was appointed for the suspension hearing, which commenced on August 14, 1974 and was to last no longer than 15 hearing days. The hearing closed on September 12, 1974, the recommended findings and conclusions of Administrative Law Judge Perlman were delivered to me on September 23, 1974, and on September 24, the parties submitted exceptions to Judge Perlman's recommended decision.

D. *Issues and controversies.* The cancellation hearing, which is expected to continue for an indefinite period, has dealt with a broad range of questions concerning Aldrin-Dieldrin's alleged deleterious effects on the environment and on human beings.[10] In contrast, the suspension hearing has been concerned solely with whether Aldrin-Dieldrin poses a cancer hazard to human beings, and whether it provides countervailing benefits.

During the hearing, counsel for both EPA and Shell characterized the issues as "cancer and corn," although Judge Perlman correctly pointed out that the benefits also included a number of other crop uses.[11] Nevertheless, in the suspension hearing record, statements of the parties indicate that the major controversy, in fact, may be narrower than "cancer and corn." Counsel for Shell declared at the beginning of the hearing: "Your Honor, in our view the issue is really cancer."[12] Even the presiding officer, who properly sought to insure that all relevant issues were addressed, stated explicitly, "I mean there is no fooling around, the major issue is cancer."[13]

E. *Legal background.* The Administrator is authorized by section 6(c)(1) of FIFRA[14] to suspend immediately the registration of a pesticide pending the outcome of final cancellation proceedings if he determines such action is necessary to prevent an imminent hazard.[15]

> * * * the function of the suspension decision is to make a preliminary assessment of evidence and probabilities, not an ultimate resolution of difficult issues.[16]

and

> The suspension order thus operates to afford interim relief during the course of the lengthy administrative proceedings.[17]

In accordance with the proposition that a suspension order is not a final determination on the merits of cancellation, but rather a temporary decision, the Agency has taken the position that it has a continuing responsibility to review suspension decisions. In his order of March 18, 1971,[18] then-Administrator William D. Ruckelshaus stated that the Agency would be prepared to reevaluate the question of suspension at any later stage in the administrative proceedings. In its most recent suspension order, in this proceeding, the Agency stated "The Administrative process is a continuing one, and calls for continuing re-examination at significant junctures."[19]

---

[4] The Administrative Law Judge noted on several occasions during the suspension hearing that the cancellation proceeding on Aldrin-Dieldrin was characterized by a fair amount of footdragging. See, e.g., Transcript 306.

[5] See Bell v. Goddard, 366 F. 2d 177, 181 (7th Cir. 1966), where an administrative action was based on reanalysis "which perhaps brought its full impact to the attention of the experts for the first time."

[6] Environmental Defense Fund v. Environmental Protection Agency, 456 F. 2d 528, 538 (C.A.D.C. 1972) [hereafter EDF v. EPA].

Note that in Nor-Am Agricultural Products, Inc. v. Hardin 435 F. 2d 1151 (7th Cir. 1970), cert. denied 402 U.S. 935 (1971), the court held that a suspension order, since it was not a final Agency decision, was not judicially reviewable under FIFRA or the Administrative Procedure Act. The Nor-Am decision was criticized in dicta in Environmental Defense Fund v. Ruckelshaus, 439 F. 2d 584, 591–592 (C.A.D.C. 1971) [hereafter EDF v. Ruckelshaus].

[7] Statement of the Reasons Underlying the Decision on Cancellation and Suspension of DDT, 2,4,5,-T, and Aldrin and Dieldrin, March 18, 1971.

[8] EDF v. EPA, 465 F. 2d 528 (C.A.D.C. 1972).

[9] The Administrator exempted those registered uses involving subsurface ground insertions for termite control, mothproofing processes using a closed system, and the dipping of roots or tops of nonfood plants.

[10] Testimony on environmental (non-human health) effects of Aldrin-Dieldrin has been presented in the cancellation hearing relating to Dieldrin residues in marine and freshwater aquatic organisms, birds, land mammals, and soil invertebrates. Because of its persistence and ubiquitous presence in nature, it is regarded as a particularly troublesome potential threat to the environment. Considerable testimony has been provided relating to its acute and chronic toxicity, transport mechanisms, bioaccumulation, and biomagnification characteristics, resistance of certain species, and various effects on the respiratory and reproductive mechanisms of fish and terrestrial life. These environmental factors, as well as other human health hazards, although not the subject of this suspension proceeding, will be carefully considered in the final Agency decision on cancellation.

[11] Counsel for Shell Chemical Company stated, for example, that "corn, that is really all we care about." Transcript. 87. See also Transcript 123, 294.

[12] Transcript 87.

[13] Transcript 92.

[14] 7 U.S.C. 136d(c)1.

[15] The Department of Agriculture has contended from the beginning of the suspension hearing that there has been an unlawful commingling of "prosecutive, adjudicative, and judicial functions required to be performed under FIFRA." (See Transcript, p. 37.) This is an interesting assertion because prior to 1970 the functions of FIFRA, including suspension, were performed by the Secretary of Agriculture Section 6(c) of FIFRA clearly states that the Administrator shall issue the notice of intent to suspend and, later, make the suspension decision.

Shell also has repeatedly alleged that unlawful *ex parte* consultations gave rise to the 2 August 1974 Notice of Intention to Suspend. I am completely convinced that any and all consultations between me and my staff which led to the decision to initiate the suspension proceeding were entirely proper and in accordance with due process requirements, administrative law and practice, and fundamental notions of fair play in the conduct of Agency adjudicatory proceedings and therefore find the assertions of USDA and Shell to be unfounded.

The function of a suspension order is not to reach a definitive decision on the registration of a pesticide, but to grant temporary, interim relief. The Circuit Court of Appeals for the District of Columbia twice has stated this view:

[16] EDF v. EPA, 465 F.2d at 537.

[17] EDF v. Ruckelshaus, 435 F.2d at 589.

[18] 18 March 1971 Order: Reasons Underlying the Registration Decisions Including Products Containing DDT, 2,4,5-T, Aldrin and Dieldrin, p. 12.

The Administrator, as noted above, may suspend when he finds that an "imminent hazard" would result during the pendency of cancellation proceedings. Section 2(1) of FIFRA[20] defines the term "imminent hazard" as "a situation which exists when the continued use of a pesticide during the time required for cancellation proceedings would be likely to result in unreasonable adverse effects on the environment." "Unreasonable adverse effect on the environment" is defined by section 2(bb) of FIFRA[21] as "any unreasonable risk to man or the environment, taking into account the economic, social, and environmental costs and benefits of the use of any pesticide."

The Circuit Court of Appeals for the District of Columbia has amplified the statutory definition of imminent hazard: "But we must caution against any approach to the term 'imminent hazard,' used in the statute, that restricts it to a concept of crises."[22]

In another case, the Court declared: The [Secretary of Agriculture] has concluded that the most important element of an "imminent hazard to the public" is a serious threat to public health, that a hazard may be imminent even if its impact will not be apparent for many years and that the public protected by the suspension provision includes fish and wildlife. The interpretations all seem consistent with the statutory language and purpose.[23]

In addition, the Administrator, in his order of March 18, 1971 specifying the criteria for determining an "imminent hazard," stated explicitly that suspension was warranted to prevent actions "which cannot be permitted to continue during the pendence of administrative proceedings. Imminent hazard may be declared at any point in the chain of events which may ultimately result in harm to the public."[24]

In a suspension proceeding, unlike a cancellation action, EPA is not required to balance possible benefits against the environmental and health risks of pesticide usage. The Court of Appeals has considered this exercise of administrative discretion by EPA and concluded: "We do not say there is an absolute need for analysis of benefits."[25]

We are not clear that the FIFRA requires separate analysis of benefits at the suspension stage. We are clear that the statute empowers the Administrator to take account of benefits or their absence as affecting imminency of hazard.[26]

The Agency traditionally has considered benefits as well as risks, however, and in my opinion, should continue to do so. The recommended decision of the Administrative Law Judge contains a lengthy discussion of the crop uses of Aldrin-Dieldrin, with their effects and alternatives. Benefits and alternatives are discussed in Part III of this opinion.[27]

In deciding to suspend because of a substantial risk of cancer in man, the Administrator is obliged to follow expressed Congressional policy of keeping carcinogenic chemicals out of the food supply. One Court has pointed out that although pesticides are not "food additives" under the Delaney Amendment, 21 U.S.C. 348(c)(3)(A), the Amendment does however, indicate the magnitude of Congressional concern about the hazards created by carcinogenic chemicals, and places a heavy burden on any administrative officer to explain the basis for his decision to permit the continued use of a chemical known to produce cancer in experimental animals.[28]

The Seventh Circuit has recently held that pesticide residues in processed foods were "food additives" within the meaning of other sections of the Food, Drug and Cosmetic Act, 21 U.S.C. 321(s).[29] But, since the Delaney Amendment does prohibit the setting of safe levels/tolerances of carcinogenic food additives, and since Aldrin-Dieldrin is present as residue in processed foods, the Administrator has a particular burden to explain a basis for a decision permitting continued use of a chemical known to be a carcinogenic in laboratory animals.

II. THE ISSUE OF THE CARCINOGENICITY OF ALDRIN-DIELDRIN

A. *General theories of carcinogenicity.* Despite the manpower and resources which have been devoted over several decades to the study of cancer, scientists are still far from agreement on the causes, nature, and even definition of cancer. In such an inquiry, where we are acting on the frontiers of knowledge,[30] we must rely on the best available evidence and interpretations and be prepared to modify our views if future scientific advances show we were in error.

A carcinogenic substance, in our opinion, is one which increases the incidence of benign or malignant tumors in exposed animals, decreases the latency period between exposure and onset of the tumor, or results in unusual tumors.[31]

The once-significant distinction between tumors and cancers, or between tumorogenic and carcinogenic substances, has lost much of its validity with the increasing evidence that many tumors can develop into cancers. Thus, for purposes of carcinogenicity testing, they should be considered synonymous.[32] Similarly, the distinction between benign and malignant tumors, while important to the individual host animal, is not a reliable indicator of carcinogenicity, for "in the thinking of most experimentalists, the induction of a benign tumor is merely a stage in a subsequent occurrence of a malignancy."[33]

This does not mean that some categorization is not useful to researchers. One recognized authority has set forth five stages of cancer development: (1) No hyperplastic lesions, (2) hyperplasia, (3) hyperplastic nodules, (4) small carcinoma (less than 5 mm), (5) large carcinoma.[34] If, for example, a pathology study found stage-four carcinoma in the exposed animals and the same number of stage-two lesions in the controls, the results would be distorted if the researcher thereby concluded that the suspected carcinogen had no effect. Such differentiation is not critical to this opinion, however, except possibly in the later analysis of certain Aldrin-Dieldrin tests on rats.

We have long known that cancer may be induced by chemicals, radiation, and even variations in the environment, but we are still not certain of the various mechanisms involved. Although four basic models have been proposed,[35] we do not have a unified model explaining the relationship between the dose and the subsequent cancerous response.

These theoretical concepts have a bearing on the Aldrin-Dieldrin issue, particularly as to the question of the existence, or non-existence, of a threshold level of carcinogenic effect. A "no-effect" level theoretically may exist, but it has not been conclusively demonstrated, and—based on the record in this case—we certainly do not know the "no-effect" level for Aldrin-Dieldrin. The lowest dose tested (0.1 ppm) still produced significant tumors in experimental animals.[36] I therefore agree with the finding of the Administrative Law Judge that "it is impossible to establish a 'safe' level of exposure of Aldrin-Dieldrin to man."[37]

---

[19] Order of August 2, 1974, at p. 4, quoting from EDF v. EPA, 465 F.2d 528 (1972).

[20] 7 U.S.C. 136(1).

[21] 7 U.S.C. 136(bb).

[22] EDF v. EPA, 465 F. 2d at 540.

[23] EDF v. Ruckelshaus, 439 F. 2d at 597.

[24] Order of 18 March 1971, supra, p. 6.

[25] EDF v. EPA, 465 F. 2d at 540.

[26] EDF v. EPA, 465 F. 2d at 538. If an analysis of benefits is undertaken, the Courts have directed that "greater weight should be accorded the value of a pesticide for the control of disease, and less weight should be accorded its value for protection of a commercial crop." EDF v. Ruckelshaus, 439 F. 2d at 594.

[27] It is, nevertheless, clear from the EPA Rules of Practice 40 C.F.R. § 164.121(g), and from the case law, that the burden of proof in establishing the safety of a pesticide product in both cancellation and suspension proceedings remains at all times with the registrant. EDF v. EPA, 465 F.2d 528, 532 (D.C. Cir. 1972); Neodane Company, Inc. v. Environmental Protection Agency, 470 F.2d 194 (8th Cir. 1972); Stearns Electric Paste Co. v. Environmental Protection Agency, 439 F.2d 584, 593, n. 34 (C.A.D.C. 1971). See also Administrator's Order of 18 March 1971.

[28] EDF v. Ruckelshaus, 439 F.2d at 596, note 41.

[29] United States v. Vita Food Products of Illinois, Inc., No. 73-1008 (7th Cir. 28 August 1974).

[30] Industrial Union Department, AFL-CIO v. Hodgson, 499 F.2d 467, 474 (C.A.D.C. 1974).

[31] The International Association for Research on Cancer (IARC) defines cancer a the induction or enhancement of a neoplasm. International Association for Research on Cancer Report, p. 9.

[32] IARC Report, p. 10.

[33] World Health Organization Reports of Cancer, EPA Ex. 40B.

[34] Statement of Melvin D. Reuber, M.D., EPA Ex. 42, p. 10.

[35] These models are the following: (1) The "one-hit" theory, derived from extensive research on atomic radiation, which holds that a carcinogenic effect may result from a single infortuitous "hit" on a single cell by some form of energy, such as a chemical. (2) The so-called logit model, derived from chemical kinetics, that there is a slow increase in response as the dose increases until finally the effect levels off when the limited number of chemical bonding sites are occupied. (3) The so-called theory of metabolic overload, which assumes that there is a threshold level in each individual, and only when that is exceeded will cancer develop. (4) The theory that everyone has a different sensitivity to carcinogenic stimuli, and that as a statistical assumption the distribution takes the form of a bell-shaped curve. It may well be that more than one theory is correct depending on many variables, but that is beyond the scope of this opinion. In any case, these four models produce very similar results within the 2-98 percent range.

[36] Shell Ex. S-3A, Tables 16 & 17.

[37] Recommended Decision and Findings of Fact and Law, Finding No. 25, p. 26.

Contrary to a wide-spread belief, it is not true that all substances are carcinogenic if introduced in sufficiently large doses. Carcinogenicity is a relatively rare phenomenon exhibited by only a few of the many hundreds of thousands of chemicals. High doses are administered in animal tests, not because the researchers seek to correlate animal response levels to humans, but because with a limited number of animals this methodology is necessary to determine gross effects. Consequently, a substance that will induce cancer in experimental animals at any dose level, no matter how high or low, should be treated with great caution.

B. *The validity of animal tests.* Most of what we know about cancer is derived from tests with experimental animals, usually mice. The response of mice to carcinogens is similar pathologically to that of man; and research laboratories, such as those of the National Cancer Institute and Shell Chemical Company, use mice extensively in their research.

Several witnesses in the hearing, such as Dr. Francis Roe of the Tobacco Research Council in London, contended that mice are not suitable test animals because they may have a high incidence of spontaneous tumors.

Although one of the five strains of mice did have a history of natural tumors, this fact alone is not significant. As Dr. Walter Heston of the National Cancer Institute pointed out:

> In testing a substance for carcinogenicity, the aim, therefore, is to ascertain whether it can significantly increase the incidence of any tumor, and the choice of strains for demonstrating this is usually not the most susceptible, nor the most resistant, but one with an intermediate genetic susceptibility.

The fact that heredity, hormones, diet, stress, and a host of other factors can influence tumors is thus irrelevant, since the experiments are designed to compare the effects of one variable—the chemical—on exposed animals otherwise subject to the same conditions.

Some witnesses also suggested that carcinogens can be species-specific—that is, a chemical substance might affect mice but not any other species, including man. This is theoretically possible. But of the thousands of compounds tested, the record indicates that this effect has been suggested for only one of them, and even this single exception has been seriously challenged. I therefore

If carcinogens are not species-specific, it logically foll.ws that the demonstration of carcinogenic effect in more than one species is not absolutely necessary for a finding of carcinogenicity.

Most carcinogens are also not organ-specific. In a survey by Dr. Tomatis of 58 compounds known to produce liver tumors in mice, 40 also induced tumors in a variety of other organs. Furthermore, chemically-induced tumors in one species need not appear in the same organ in another species. Thus, a carcinogen which induces liver tumors in mice might, for example, produce mammary cancers in rats and lung tumors in men.

C. *Carcinogenicity of aldrin-dieldrin in mice.* There is no dispute that Aldrin-Dieldrin significantly increases the incidence of liver tumors in five different strains of mice. There is overwhelming scientific data supporting this fact, and the registrants have now conceded this point: The main result from the initial analysis was that in all Studies there was a highly significant dose related increase in the liver tumors.

The IARC has concluded that: Dieldrin was tested by the oral route only in mice and rats. The hepatocarcinogenicity of Dieldrin in the mouse was demonstrated and confirmed in several experiments, and some of the liver tumors were found to metastasize. A dose-response effect has been demonstrated in both sexes with an increased incidence in the females at the lowest dose tested, 0.1 ppm in the diet.

Shell's own test results confirm the above conclusions. In exposed groups, all three strains of mice in the seven tests had a high increase in the incidence of liver tumors. The first two tests (Study 1 and Study 2.1) are the most meaningful because the test populations were much larger than in the other tests and the dose levels ranged low enough so that acutely toxic effects did not interfere with the development of slower tumors. The mice tested were also from inbred, outbred, and hybrid strains.

The test results show that the increase in the incidence of tumors was dose-related, although at doses above 10 ppm this relationship was diminished because of interference by acutely toxic effects. At the lowest dose level tested, 0.1 ppm, there was an increase in benign and malignant tumors. Those that did develop had a greater tendency to spread to other sites in the body and especially to the lungs.

Aldrin-Dieldrin shortened the latency period in the development of tumors in both sexes. In one test measuring the effects of limited exposure, the compound increased the incidence of tumors after exposures as short as two weeks; the effects were even more pronounced after one mouth of exposure.

The incidence of malignant liver tumors was statistically significant in almost every test Shell performed. This elevated incidence of malignancy is particularly important because these strains of mice were especially resistant to malignant liver tumors. The incidence of malignancy in female controls was almost nil and in males it was quite low.

Exposure to Aldrin-Dieldrin and DDT apparently has synergistic effect on the development of tumors. Mice fed 50 ppm DDT had some increased incidence of tumors. However, when mice received a diet of 5 ppm Aldrin-Dieldrin in addition to 50 ppm DDT, the incidence of tumors increased sharply: Males had 4 times and females 8 times as many malignant tumors as those exposed only to DDT. Dr. Reuber has concluded,

It certainly is clear from these observations that Dieldrin and DDT have additive effects when it comes to carcinogenicity. Further, the evidence indicates that Dieldrin is primarily responsible for this important effect. Using the 50 ppm group as the controls the carcinogenic effect of the combined

---

EPA Ex. 48, pp. 7–8; EDF Ex. 33, pp. 20–21; Transcript 9222–3, 9285, 9580–32, 9642–44.

For example, a chemical that produced one cancer in every thousand human beings would be a great tragedy indeed, but very few laboratory tests include as many as a thousand animals, and even then one incidence might be disregarded as chance.

EDF Brief, V–66 et seq.

EPA Ex. 40D. The official Mrak Commission Report on Pesticides has stated: The use of nonrodent species * * * has now been substantially dropped. A suitable, practical nonrodent species would be useful, but it is not available at this time. Carcinogenicity tests of food-borne pesticides require routine lifetime feedings of chemical compounds. While dogs have been employed for tests of carcinogenicity, with noteworthy success in selected cases (bladder carcinogenicity of aromatic amines), the requirement of lifetime feeding makes this species too expensive, in terms of time and funds, to be employed routinely. EPA Ex. 40F.

Although during the hearing Shell argued that mice were an inappropriate test species, one of the reasons for the relative shortage of Shell data on other species is that Shell preferred to use mice in its own cancer experiments.

Shell Ex. 12, pp. 20–22.

In any case, three of the four remaining mice strains were especially resistant to spontaneous liver lesions (these were the strains used by Shell) and the fourth had only average susceptibility. This will be discussed in more detail in the next section.

EPA Ex. 46, p. 11.

EPA Ex. S–11.

The exception, according to Shell, is Phenobarbitone, which is supposedly carcinogenic in mice, but not in man. Shell Ex. 14, based on Dr. Clemmesen's study of epileptics. Arsenic may have the obverse effect, but the mice tests are still not conclusive. See Perlman, Recommended Decision, p. 41.

Dr. Schneiderman has been quite critical of the Clemmesen study and contends that a mathematical re-analysis of his results is "consistent with the possibility that the anticonvulsants which the epileptics received increased the risk of liver cancer, perhaps two or three fold." EPA Ex. 10, *p*. 9.

will rely on the conclusion of such organizations as the International Association for Research on Cancer, which have rejected species-specificity as unsubstantiated.

EPA Ex. 40–H. Even if species-specificity does exist, it has not been demonstrated for Aldrin-Dieldrin by the record in this case.

An HEW Advisory Panel has recommended that a finding of carcinogenicity be made when a substance is "judged positive for tumor induction in one or more species * * *." EPA Ex. 40F, p. 468.

EPA Ex. 50–H; see also EPA Ex. 40–B, Annex 1.

EPA Ex. 48, p. 15. Note, however, that in the recent Polyvinyl Chloride episode, both mice and men developed rare angiosarcoma of the liver.

Shell Ex. S–2A, p. 2.

EPA Ex. S–17, pp.143–44.

Study 1's population was over 1000 with dose levels of .1, 1, and 10 ppm. Study 2.1 had a population of 400 and five dose levels of 1.25, 2.5, 5.0, 10 and 20 ppm. Note that Dr. Nathan Mantel has testified that Shell's method of analysis is an adaption of one he developed, and he criticizes Shell for failing to apply his method correctly. He states that their analysis is insensitive to patterns and consistencies and the effects of competing toxicity at high dose levels. Because of its shortcomings, Dr. Mantel feels Shell's analysis is "almost guaranteed to give non-significance for even the strongest carcinogen". EPA Ex. S–21, pp. 2–3.

Shell Ex. S–3A, Table Data 1, Table Data 2; Transcript 986.

Shell Ex. S–3A, Table Data 1.

Shell Ex. S–3A, Table Data 1, Table Data 3.

EPA Ex. 50, pp. 12, 13; EPA Ex. S–1, p. 9.

EPA Ex. 43–E, Table 8.

feeding of Dieldrin and DDT is very highly significant by statistical analysis.[61]

The World Health Organization has recognized that in exposed mice there is an increased risk that liver tumors will spread to the lungs.[62] Shell's test results have confirmed this, for at least two of their experiments demonstrate a statistically significant increase of lung tumors for both sexes. Some increase in lung tumors was observed in almost all their tests. Dr. Gross has testified that the results of the first study:

> * * * leave little room for doubt that Dieldrin at either 0.1 ppm or both the 0.1 and 1 ppm levels can elevate the incidence of tumors at sites other than the liver (particularly in the lung) and that this elevation is highly significant in either males or females or in both sexes.[64]

D. *Carcinogenic effects on rats.* Rats have been used less frequently than mice as test populations. The quality of the tests has varied widely, and the results have not been uniform. For those reasons the Administrative Law Judge concluded.

> We are hesitantly unwilling at this time to find that Dieldrin is conclusively a carcinogen in the rat although there are indications that this is so especially when the chemical is tested at the lower dosages * * * we are certain, nevertheless that the findings in the rat cannot be described as negative. (Emphasis in original.)[65]

This caution is warranted by the serious deficiencies in the available rat tests. However, it is my conclusion, following an intensive re-examination of the statistics and testimony presented in the recent hearing, that there is a strong probability that Aldrin-Dieldrin is a carcinogen in rats as well as mice.[66]

The two series of tests conducted by the Food and Drug Administration (FDA) are useful for determining the effects of Aldrin-Dieldrin on rats. Exposed rats had a markedly increased incidence of liver and other tumors, which was especially noteworthy because the tested strains had a low rate of natural liver tumors.

The rate doubled for rats exposed to Aldrin and increased by one-third for those exposed to Dieldrin. A no-effect level was not observed. The liver to body weight ratio increased, and at high doses there were serious enlargements of the liver. After six months, a dose-related decrease in survival rates was observed. In over 90 percent of the rats dying at high dose levels, lesions were present.[67]

After reviewing the FDA tissue slides, Dr. Reuber confirmed the increase in tumors. He found that at the low dose levels (1.5–10 ppm) there was a low incidence of liver tumors but an increased incidence of tumors in other organs. At higher doses, there was a higher incidence of liver tumors. This incidence of tumors more than doubled at both low and high dose levels. While no liver tumors were observed in controls, 18% of the rats at high dose levels had liver tumors.[68]

These results are confirmed by Shell's own test results, which show that almost twice as many exposed rats had tumors and the liver to body weight ratio among female rats increased at low doses.[69]

E. *Tests on other species.* Aldrin-Dieldrin has also been tested in species other than the mouse or the rat. Almost all these tests have been on dogs and monkeys and are not very useful, due to their small populations and test durations shorter than the cancer latency period.

There have been three dog experiments. The populations have been small, ranging from 1 to 5 animals per dose level, with a duration not exceeding two years. In spite of these obvious test inadequacies, after two years of exposure dogs had diffuse hyperplasia of the liver which "was such that over a period of several years the dogs could have developed carcinoma of the liver."[70] In commenting on the weaknesses of the dog tests, Dr. Saffiotti has stated that an acceptable test:

> * * * would require a duration of at least ten years to come close to the age at which tumors could begin to be found. For example, benzidine, a potent carcinogen for the urinary bladder in man as well as dogs, took about seven years to produce its first tumor in dogs. The number of animals needed for statistical evaluation of tumor incidences in treated and control groups is dependent on mathematical and not zoological criteria, so that there is no reason to accept experiments on groups of one or two or five dogs any more than there is to accept experiments in one or two or five mice. In conclusion, these dog studies are completely and utterly inadequate as carcinogenesis tests and should be totally discarded in the consideration of the carcinogenic response to Dieldrin.[71]

There has been only one monkey test, which had five monkeys at each of five dose levels, and six controls. The test duration was about six years. During that time there was some evidence of microenzyme induction, but there were no observations made on tumors.[72]

Dr. Saffiotti has stated that: However, as in the case of dog studies, the number of animals used and the duration of the test for only approximately one-fourth of the expected lifespan of this species, make this study totally inadequate as a carcinogenesis test.[73]

F. *Extrapolation of animal data to man.* The ultimate issue in this suspension proceeding is whether Aldrin-Dieldrin is carcinogenic in man. Because man's response to carcinogens is similar to that of rodents, the finding that a substance is carcinogenic in experimental animals indicates that it poses a similar risk to man. Dr. Heston has testified:

> Knowing this, and knowing the general biological similarity of mice and other mammalian species, including man, we can reasonably expect that in a population of human beings exposed to Aldrin-Dieldrin, cancer of some kind will occur in some individuals, and these individuals will not have been afflicted in the absence of these compounds * * * The human population is so much more genetically diverse than any laboratory animals, that if a chemical has been shown to be carcinogenic by a significant induction in any laboratory strain of mammal, we can reasonably expect that at least certain human beings would also respond to the chemical by developing some kind of neoplasm.[74]

The strongest position for the registrant was taken by Dr. Don Stevenson, Director of Shell's Tunstall Laboratory, who testified that evidence of human carcinogenicity is only sufficient when five criteria are met:

1. The exposed animals experience a higher incidence of tumors.
2. Tumors develop in more than one species.
3. The development of these tumors can be proven to be compound-related.
4. The animal has proven to be an adequate model for extrapolating to man.
5. Human data is available proving at least one incidence of cancer that is compound-related.[75]

It is no exaggeration to say that Dr. Stevenson's demands are practically impossible to meet.[76] Our knowledge of cancer mechanisms is still imperfect and it may take many years before we understand the mechanisms with certainty. Furthermore, epidemiological studies are difficult or impossible to conduct on the effects of Aldrin-Dieldrin.

It is the carcinogenic effect of Aldrin-Dieldrin, not the mechanism that concerns us here. The evidence is conclusive that Aldrin-Dieldrin is carcinogenic in mice. It has produced statistically significant compound-related benign and malignant tumors in the livers of five different strains of mice. It also significantly increases the incidence of lung tumors. This evidence of carcinogenicity is supported by additional, although not definitive, evidence that Aldrin-Dieldrin has increased the incidence of tumors in rats. Dr. Upton, a recognized cancer expert, has testified:

> In safety testing of carcinogens today we are concerned with one question:
>
> Does exposure to the test agent result in a significant induction of tumors in exposed populations as compared to controls? If so, then the test agent has elicited a carcinogenic response and must therefore be considered potentially hazardous to human health. Whether the agent actually is a *sine qua non* of the observed response or merely enhances a virus or some other factor found in the host animal is irrelevant unless and until we know that similar factors are not also found in man. Until we have such knowledge, we have no basis on which to make distinctions between "carcinogens"

---

[61] See Shell Ex. 3–A Tables 16 and 17. For example, the Shell Study 2.2 shows a significant increase with 1.22% of the controls and 20.66% of the exposed mice developing malignant tumors. This has a very low chance probability of .000000048. Almost three times as many of the treated mice had benign or malignant tumors as did the controls (*EPA Ex. S–1, p. 18*). However, Shell contends that even though the increase in lung tumors is very high, this increase is incidental to the development of liver tumors and therefore, they reason, it cannot be proven to be caused by Aldrin-Dieldrin.

[62] EPA Ex. 42, p. 26.

[63] Shell Ex. S–4, p. 20.

[64] EPA Ex. S–9, p. 29. Dr. Gross found significant increases in lung tumors, regardless of whether liver tumors were present, and a decrease in the latency period. Over three time as many (77.8%) exposed females developed lung tumors within two weeks as did the control females. (EPA Ex. S–1, p. 9.)

[65] Recommended Decision, pp. 56–57.

[66] This determination that Aldrin-Dieldrin is probably carcinogenic in two species is helpful, but not absolutely essential, to a finding of imminent hazard, as the data on mice is sufficiently strong to justify a finding of carcinogenic risk.

[67] EPA Ex. 33.

[68] EPA Ex. 42. At low doses female rats had an especially high incidence of liver tumors. At high doses the incidence of liver tumors was not as pronounced as should be expected because the rats died from the toxic effects before tumors could fully develop.

[69] Shell Ex. S–13.

[70] EPA Ex. 42, p. 38.

[71] EPA Ex. 40, p. 33.

[72] Transcript, 1082.

[73] EPA Ex. 40, p. 32.

[74] EPA Ex. S–11, 5 & 7.

[75] Transcript 537–555.

[76] Dr. Stevenson's position on the necessity of proof for two species is particularly interesting, since as Director of Shell's Laboratory, he feels that it is no longer fruitful to do research on rats. Furthermore, in spite of Shell's strong position on the necessity for human data, the Registrant is no longer studying Aldrin or Dieldrin's effects on man.

and "co-carcinogens" and "causative agents" versus "enhancing agents".

Given this lack of knowledge concerning mechanisms, I believe that a carcinogenic reaction in any species of test animal must be considered sufficient to describe the test compound as a carcinogen and so a threat to human health. I consider that a similar reaction in a second mammalian species is a confirmation of the carcinogenicity of the test agent but it is not necessary before a finding of carcinogenicity and threat to human health can be made; and negative results in a second or even third species of test animal do not in my mind establish that the test agent is not a threat for human beings. Given the variation in human susceptibility to carcinogens, I believe it unreasonable to ignore a finding of carcinogenicity in any mammalian test species when considering possible effects on human health.[77]

G. *Body burden and intake.* There is a conclusive evidence that residues of Aldrin-Dieldrin are present in virtually every member of the U.S. population. An EPA Human Monitoring Study has established that in 1971, 99.6 percent of the persons sampled had Aldrin-Dieldrin residues in their adipose tissue.[78] The compound also has been found in the blood samples of 69 percent of the population tested.[79]

In the environment, Aldrin-Dieldrin is most frequently present in food crops, and the consumption of food has been man's principal exposure to the compound. The FDA's Market Basket Surveys have shown that the compound is present most frequently in dairy products, meat, fish, poultry, and fruits. Residues were found in 83 percent–96 percent of these products. These particular commodities contained almost all of the Aldrin-Dieldrin residues found in the Market Basket Surveys. Although the levels of these residues has fluctuated somewhat, there has been no significant decline in their presence in recent years.[80] Another EPA Monitoring Study has found Aldrin-Dieldrin residues in 85 percent of the air samples taken.[81]

There is inconclusive evidence on the relationship between the intake of Aldrin-Dieldrin and body burden levels. However, it appears that the longer the exposure, the higher the tissue level.[82] The concentrations of Aldrin-Dieldrin in the adipose tissues of the general population have been found to be comparable to the levels in mice exposed to 0.1 ppm of the compound.[83] After exposure, species eliminate the compound from their systems at different rates. Rats excrete the compound with a half life 4 to 6 times as fast as mice and 13 to 26 times as fast as humans.[84]

We are uncertain as to the precise effect of Aldrin-Dieldrin on fetuses and infants[85] but are concerned because their intake levels can be over six times the so-called Acceptable Daily Intake (ADI) level. Breast-fed babies are particular susceptible, as virtually all human milk has considerable Aldrin-Dieldrin residue.[86]

H. *Epidemiological studies.* Epidemiological studies on the carcinogenicity of Aldrin-Dieldrin have been inadequate and inconclusive. Although it may be true that all known human carcinogens have only been identified through epidemiological studies, the identification of the carcinogenic effects of Aldrin-Dieldrin through such studies would be difficult because there is no member or segment of the human population that has not been exposed to the compounds.[87]

Shell has agreed that their epidemiological study does not prove that Aldrin-Dieldrin is non-carcinogenic.[88] Their tests detected no effect among the subject population, even though some mortality and morbidity was observed.[89] However no conclusion can be drawn from these results because the test does not meet basic standards of acceptability.[90] The test population was too small, the period of exposure was too short, and the medical observation periods were not long enough to approximate the expected latency period of at least 20 years for Aldrin-Dieldrin.[91]

III. THE USES, BENEFITS AND ALTERNATIVES FOR ALDRIN-DIELDRIN

A. *Relevance of the benefits issue.* In view of the foregoing health risks, do the benefits of Aldrin-Dieldrin justify its continued use? A related question is whether alternative pest controls exist and will be available for the 1975 growing season. The "availability" of alternatives assumes several factors, including timely registration, effectiveness, adequacy of supply, safety, and economy.

The following integrated discussion pertains only to the possible effects of suspending Aldrin-Dieldrin for the duration of the cancellation proceeding.

Since Aldrin-Dieldrin has been found to be carcinogenic in mice and probably carcinogenic in rats, and to present a high risk of cancer to man, it is arguable that any use of Aldrin-Dieldrin, however significant or beneficial in social or economic terms, cannot be justified, even for the limited period of time until the completion of the cancellation proceedings.

As indicated in part I of this opinion, however, it is appropriate that the possible benefits of Aldrin-Dieldrin, or the absence of such benefits, be considered in this proceeding. Nevertheless, it is apparent that any benefits attributable to Aldrin-Dieldrin must be of a high order to affect the findings on carcinogenicity.[92]

The following sections, therefore, analyze the major points raised in the hearing relating to uses, benefits, and alternatives, to determine whether any of these benefits justify the continuing risk.[93]

B. *The significance of aldrin-dieldrin uses on corn.* During the 1950's and 1960's, Aldrin-Dieldrin became the leading insecticide for the control of several corn pests.[94] From that period of widespread application, Aldrin-Dieldrin use has declined to only about 8% of the nation's total corn production acreage.[95] Changes in corn production over this period gradually have reduced reliance on chemical insecticides to sustain high crop yields. These changes resulted from a variety of factors, including the benefits of new hybrids, the availability of synthetic nitrogen fertilizer, and advanced farm management practices.[96] These changes in cultivation also have helped to reduce corn insect populations. Crop rotation practices and the increase in soybean production in the last decade have eliminated some of the favored insect nesting areas.

	Acres (millions)	Percent of U.S. corn growth with aldrin
1971	9.4	12.9
1972	7.5	11.6
1973	7.4	10.6
1974 (preliminary)	5.9	7.8

---

[77] EPA Ex. S–19, pp 4–5.

[78] EPA Ex. 36, Tables I and II, EPA Ex. S–15. Other years deviated from these results insignificantly. Individual samples varied widely from the mean of .27 ppm, with some as high as 116.55 ppm.

[79] EPA Ex. 36, Table III.

[80] EPA Ex. 38A, Table I and II. The average intake in 1973 was .002 mg/day. (.00003mg/kg/day). The study has been criticized for having too small a sample and for poor analytical methods; its figures are unquestionably low. (EPA Exhibit 30). (Tr. 15,281). Although the absolute intake values may not be known precisely, their relative values are evident from the study.

[81] EPA Ex. 37. There is evidence that this figure may be low due to absorption in lungs and clothing.

[82] EPA Ex. 8Q.

[83] Transcript 597–598. Thus it may diminish the relevance of placing the emphasis on the intake rather than the tissue level.

[84] Transcript 599.

[85] No tests have been performed on infants in any species to determine their level of susceptibility. However, some scientists consider it to be quite high.

[86] Transcript 32. The ADI (.0001 mg/kg/day for Aldrin-Dieldrin) was established in 1966 long before the most meaningful tests were run on mice proving the carcinogenic effects of Aldrin-Dieldrin. Although the ADI is defined as a no-effect level, it is actually a threshold level based on a rat study at 0.5 ppm in which exposed rats experienced liver changes (Transcript 769) (Shell Ex. 4, p. 16).

[87] Many compounds induced tumors of an unusual type, which facilitated the identification of the carcinogens. In other cases, the tumor manifested itself in a distinct population before there was a suspicion of carcinogenicity so it was easy to relate the effect back to the cause. These situations do not apply to Aldrin-Dieldrin. As Dr. Gross testified: Even if Aldrin and Dieldrin were to pose a very significant danger to humans, really an impressive, even a catastrophic one, we would never know this. (Transcript 323)

[88] Shell S–4, p. 31; Transcript 506.

[89] There was one death in the high exposure group of stomach cancer, but this death was considered insignificant. In the same high exposure population, one worker developed a tumor during exposure and another, leukemia. It is Shell's position that the test showed no incidence of enzyme induction, liver injury, or the presence of alphabeta protein. From this, they seem to imply that this is evidence that Aldrin-Dieldrin is not carcinogenic. However, as Dr. Farber has stated, cancer can develop without these symptoms. (See EPA Ex. S–15). Dr. Van Raalte takes the lowest level of exposure in this test, which is 175 x the ADI, and adopts it as a no-effect level. (Transcript p. 681.)

[90] EPA Ex. S–17, p. 11.

[91] EPA Ex. S–10, p. 6. The average occupational exposure was 6.6 years; the average observation period, 7.4 years; and the average age, 47.4. There were 169 men who were exposed at high dose levels. (Shell Ex. S–4).

[92] EDF v. Ruckelshaus, 439 F.2d 584, 596 at note 41.

[93] This evaluation does not necessarily mean that the final decision in the cancellation proceeding will be the same, for a wider range of topics (including other health effects) and additional evidence on both risks and benefits will be considered in those hearings.

[94] See EPA Brief, pp. 181–183, citing the successes of Aldrin-Dieldrin and Heptachlor in the 1950's (Decker Shell Ex. 12). Sales of Aldrin peaked in 1966 (Shell Ex. 111, p. 38), and for corn use in Illinois in 1967 (EPA Ex. 60, p. 9):

[95] See EPA Brief, p. 207, citing USDA figures (Shell Ex. S–17A) showing Aldrin use declined from 13.4 million acres in 1966 (20.2% of U.S. corn acres planted) to 7.5 million acres in 1971 (10.2%). The Doane survey shows a continuing decline since 1971 as follows: (EPA Ex. S–16, p. 3).

[96] See EPA Brief, p. 183–188, citing testimony by Dr. Petty (EPA Ex. 60, p. 2–3) and Dr. Fairchild (Shell Ex. S–16, p. 12).

Reproduced from best available copy.

The recent hearing strongly indicated that the incidence of significant infestations of the major corn soil insects today is extremely low. For example, Dr. Petty testified that there were no major corn insect problems in Illinois in 1972,[97] and Dr. Turpin stated that wireworm and cutworm populations in Indiana were at a very low level.[98] A research team in Iowa, according to Dr. Owens, found less than ten wireworm incidents in 1972 and 1973, only two significant black cutworm infestations, and no white grub problems.[99]

Corn farmers, nevertheless, continue to use large quantities of Aldrin-Dieldrin as prophylactic or "insurance" protection against potential pest damage, even where an actual threat of economic injury is not specifically determined. In other words, the pesticide is used even if there is no indication that it is needed.[100]

Aldrin-Dieldrin may no longer be as effective or as necessary in controlling these corn pests as has been claimed or as has been assumed by its users. These doubts are due to the developing resistance to Aldrin-Dieldrin by some corn pests, the current low incidence of corn insect infestations on both treated and untreated acreage, and the lack of recent data on the pesticide's continuing potency.[101]

C. *Alternatives to aldrin-dieldrin for corn.* The most important corn insect pest is the rootworm. Since 1962, it has been known that rootworms were becoming resistant to Aldrin-Dieldrin,[102] and it is now established that two of the three types of corn rootworms are resistant.[103] In view of the fact that other insecticides are available to control rootworm,[104] and the fact that Shell apparently does not place major importance on the use of Aldrin-Dieldrin for rootworm control,[105] corn rootworm control does not present a convincing need for the use of the compound in 1975.

The black cutworm generally inhabits low-lying, poorly-drained river bottom land, heavy soils, and the low, wet areas of upland fields. The loss in crop stand and yield from cutworm infestation can, on occasion, be substantial.[106]

Wireworm is the third major corn pest. It appears to be associated with cropping patterns where corn is grown after sod or pasture, and is primarily a problem only in first-year corn. Thus, it is generally not a problem after the first year or where soybeans and corn are rotated.

The record indicates that registered alternatives are available for all these pests, although Shell disputes their effectiveness. For corn rootworms, the alternatives include Diazinon, Mocap, Thimet, Furadan, Dasanit, and others. Most of these, and other chemicals, also are registered as effective for control of wireworms. Alternatives registered for cutworms on corn include Carbaryl, Dylox, and Diazinon, with registration pending also for Furadan.[107]

Minor soil insects, such as white grubs, seed corn beetles, seed corn maggot, grape colaspis, corn billbug, Japanese beetle, Asiatic Garden beetle, corn root aphid, corn field ant, flea bettle larvae, or clover root borer, do not pose any significant economic threat to corn production.[108] Where white grubs do exist, some control can be obtained by organophosphates, such as Malathion, or carbamates used to control rootworms or wireworms.[109]

The record further indicates that these alternative pesticides should be available in sufficient quantities for the 1975 season, especially since a pound-for-pound substitution for Aldrin-Dieldrin is neither necessary nor desirable.[110] Shell's own estimates of available supplies indicate significant increases in production of some alternatives and continued high production levels for most others.[111]

D. *Projections of corn crop reductions.* Corn production in the United States is of considerable importance to the nation's economy. Fortunately, the suspension hearing record indicates that the macroeconomic impact of the proposed suspension order would be almost negligible.

The most reasonable projection[112] was the study conducted by Dr. Delvo of the U.S. Department of Agriculture, who predicted that corn crop reduction could amount to as much as 0.4% of expected production.[113] Even this estimate may be considerably inflated, as EPA witness Dr. Aspelin, pointed out, because it assumes a level of wireworm and cutworm infestation considerably in excess of current field estimates.[114]

A second study was conducted for Shell by Doane Agricultural Service. The farmers' loss estimates were ten times as high as the Delvo prediction, plus another five times due to a claimed shifting of production from corn to another crop.[115] This projection seems somewhat high, considering that Aldrin-Dieldrin is used on less than 8% of the nation's total corn crop. Shell has conceded that "because of certain methodological problems and the questions concerning the ability of farmers to make estimates, Mr. Wilkin's estimate may be too high."[116]

A third study, conducted in 1973 by Dr. Freund, assumed the simultaneous unavailability not only of Aldrin-Dieldrin but also of Chlordane and Heptachlor, and consequently projected losses in the range of 0.7 to 1.6 percent. This "very rough study," which was clearly "tentative and preliminary," cannot constitute a reliable basis for a conclusion on macroeconomic impact.[117]

It is possible that there may be no crop reduction at all due to the lack of Aldrin-Dieldrin. For fields with significant insect damage to the young plants, crop loss can be greatly reduced by immediate replanting and treatment with an alternative pesticide.[118] This is a common practice and may be less expensive overall than extensive prophylactic treatments used by many farmers.

I, therefore, concur in the finding of Judge Perlman who, after reviewing the above studies and projections, concluded: "On the basis of the foregoing, we cannot find any major economic or social benefit resulting from the use of Aldrin on corn in the context of overall effect of its unavailability for such use."[119]

E. *Citrus uses of aldrin-dieldrin.* Although the benefits portion of the suspension hearing dealt almost solely with corn, there are a number of "minor" uses for which the Department of Agriculture (USDA) contends that Aldrin-Dieldrin is essential. The most important of these is the use on various citrus pests in Florida.[illegible]

---

[97] EPA Ex. 60A, p. 109; Transcript (Cancellation) 11141–44, 1135; EPA Ex. 60, p. 7.

[98] Transcript (Cancellation) 11482, 11492, 11561; Indiana Survey for 1972–72, EPA Ex. 61, pp. 22–26.

[99] EPA Ex. 71; see also the testimony of Dr. Stockdale, Transcript (Cancellation) 22636, 22783, 22974.

[100] See, e.g., testimony of various farmers that Aldrin has been used as insurance against insect attack (Garst, Transcript (Cancellation) 284; Decker, Transcript 162; Kirk, Transcript 22, 550; EPA Ex. 61, pp. 34–35).

[101] See testimony of Dr. Petty, EPA Ex. 61, p. 36; Transcript 11560; EPA Ex. 60, p. 7; Transcript 11398; and Dr. Sechriest, Transcript 11794; EDF Brief III A2, pp. 7–9.

[102] See EPA Brief, pp. 188–193, citing various sources concerning what appears to be a conceded fact in these proceedings. EPA Ex. 68, pp. 3–4; Transcript 11074; EPA Ex. 60, p. 3.

[103] The two resistant species are Western and Northern corn rootworm. See Recommended Decision, p. 83.

[104] Among the registered and recommended alternatives listed by EPA are Furdan, Thimet, Dasamit, Dyfonate, Diazinon and Mocap. Counter has a temporary use permit and is expected to be finally registered for rootworms and wireworms by late 1974. EPA Brief, p. 193.

[105] Shell apparently concedes that Aldrin-Dieldrin is not an efficacious treatment for rootworms. No arguments for its use on rootworms are set forth in Shell's Brief No. V in the cancellation proceeding or in their post-hearing brief in the suspension proceeding. The Chief Administrative Law Judge specifically found corn rootworm control not to be a consideration with respect to the need for Aldrin-Dieldrin. Recommended Decision, p. 83.

[106] Sechriest, EPA Ex. 63G, p. 3.

[107] EPA Brief, Table, pp. 176–177. Heptachlor and Chlordane also are registered and effective for certain applications. The Agency does not consider them safe alternatives, even though the scientific case against them is not yet as complete as that against Aldrin-Dieldrin. (See also p. 39, note 1).

[108] See testimony of Dr. Turpin (EPA Ex. 61, p. 40); Transcript (Cancellation) 11141, 15330–3.

[109] See EPA Ex. 60T, p. 88, showing some control of white grubs with band applications of Dasanit, Dyfonate, Diazinon, Thimet, and Furadan.

[110] Hopefully, one result of this decision will be to reduce unnecessary "insurance" applications of insecticides and to limit their usage to situations where they can prevent significant economic injury.

[111] Production of Furadan, Dyfonate, and Mocap, among others, will be substantially increased next year. Shell Brief, pp. II–8 to II–13; see also EPA Brief, p. 206.

[112] Judge Perlman described the Delvo Study, despite certain problems, as "the on economic study offering some reliance." Recommended Decision, p. 79.

[113] Shell Ex. S–17A.

[114] Ibid.

[115] Shell Ex. 165.

[116] Shell Brief, p. II–19. The hearing examiner concluded, "We totally reject the Doane Agriculture Service, Inc., special survey and projections of loss * * *. On its face, it is patently exaggerated, employs 'double counting compounded,' is based on a small sample from which averaging projections are made and elicited the views of Aldrin users who would not in reality know with any precision the effects of the absence of Aldrin and who, it seems to us, would demonstrate a bias." Recommended Decision, p. 79.

[117] Recommended Decision, p. 79. Even though the EPA staff believes that Heptachlor and Chlordane pose a "substantial question of safety" sufficient to initiate the cancellation process, and therefore does not recommend them as alternatives, as a factual matter these compounds will be available for the 1975 growing season. The fact that the Agency has not yet initiated administrative proceedings on Heptachlor and Chlordane is not relevant to the hazards of Aldrin-Dieldrin. It would be extremely irresponsible to refrain from banning the use of one carcinogenic compound because another compound might also have carcinogenic effects.

[118] Shell Brief, p. 10.

[119] Recommended Decision, p. 81. Regardless of minimal economic impact at the national level, it is always possible that some individual farmers may be more disadvantaged than others by the suspension of a particular pesticide. It is my interpretation of the FIFRA, however, that these burdens on individual farmers must be severe and widespread to justify exposing the entire population to a demonstrated carcinogen.

Aldrin-Dieldrin and other insecticides for control of the Fuller Rose Beetle are only used on 1–5% of citrus acreage in Florida.[illegible] Even in the Indian River area of Florida, soil insecticides have been applied only to some 20% of the acreage.[illegible] As with corn, Aldrin-Dieldrin has been used extensively for "insurance" protection on citrus, where actual economic risk has not been specifically determined.[illegible]

Other Florida citrus pests include the blue-green citrus weevil and the sugar cane root stalk borer weevil (diaprepes), but these are very limited problems, both geographically and in magnitude. Only about 1% of Florida citrus acreage is subject to weevil infestation, and of this Aldrin-Dieldrin is used on somewhat more than one-half.[illegible]

Although fewer acres of citrus than corn are treated with Aldrin-Dieldrin, the rate of application per acre is considerably higher. Whereas the rate on corn is one to two pounds per acre, the rate on citrus is five pounds. These "minor" uses on Florida citrus, therefore, account for 120,000 pounds of Aldrin-Dieldrin a year.[illegible]

Alternative foliar sprays and organophosphate soil insecticides, as well as cultural practices, are available to control all of the above minor pests. In California, which does not recommend Aldrin-Dieldrin for control of the Fuller Rose Beetle, effective results have been achieved with Malathion, Sevin, and Carathion, Furadan (in split applications), Guthion, Diazinon, and Lannate are also available alternatives.[illegible]

It does not appear, therefore, that Aldrin-Dieldrin use on citrus will be a critical need in the 1975 season.

F. Seed treatment uses of Aldrin-Dieldrin. Aldrin-Dieldrin is used in seed dressing for many types of grain, fruit, and vegetable seeds as a prophylactic measure.[illegible] Normally, only two to four ounces of dressing per 100 pounds of seed is applied, either in commercial seed preparations or by the farmer during planting.[illegible] This amounts, however, to 130,000 pounds of persistent Aldrin-Dieldrin entering the environment per annum.[illegible] This hazard is unnecessary, for alternative seed dressings are available: Diazinon, Lindane, and BHC (in Canada) are used effectively for this purpose. Proper cultural practices also can reduce the need for seed treatment.

There is, therefore, little or no evidence that Aldrin-Dieldrin seed dressing is needed to prevent significant social or economic injury.

G. *Other minor uses of Aldrin-Dieldrin.* Many other uses of Aldrin-Dieldrin, including Puerto Rican pineapples, sugar cane, and bananas, onions grown in the Tulelake Basin of Northern California, strawberries in Oregon and Washington, the USDA's quarantine program,[illegible] cranberries, and nursery stock, are defended by the USDA in this suspension proceeding. Registered alternative insecticides are available for these uses during the period required for the completion of the cancellation proceeding.[illegible] Registration of additional alternative insecticides is pending.

With respect to these other uses, there is no basis on the record to conclude that significant social or economic injury to the nation or to individual growers would result from the suspension of Aldrin-Dieldrin.

IV. CONCLUSIONS

1. Based on the testimony of record in the suspension hearing and the considerations set forth in Part II of this opinion, I have concluded that the continued use of Aldrin-Dieldrin during the time required to reach a final decision in the cancellation proceeding would be likely to result in unreasonable human health risks and, therefore, that an "imminent hazard" within the meaning of section 2(1) of FIFRA would result during the pendency of the cancellation proceeding.

2. I have concluded further, based on the testimony of record and the considerations set forth in Part III of this opinion, that there are no countervailing benefits resulting from the registered uses of Aldrin-Dieldrin that outweigh the human health risks identified, and that, in any event, alternative registered and recommended pesticides do exist and will be available for use in the 1975 growing season to provide effective pest control.

3. It should be emphasized that these conclusions are not dispositive of other risk and benefit issues and considerations pertaining generally to the cancellation proceeding. In particular, it should be noted that the fact that a sufficient need has not been demonstrated for exempting any of the minor uses (i.e., other than corn) from this suspension action should not be interpreted as conclusionary in terms of the cancellation proceeding. Further evidence and further consideration in the cancellation proceeding may show that risks associated with some of these minor uses approach *de minimus* levels and would not outweigh the possible benefits. It also is possible, in the context of the cancellation proceeding, that further evidence and consideration might warrant a different conclusion regarding Aldrin-Dieldrin use on citrus or seed treatment. Such conclusions, however, cannot be reached on the basis of the suspension hearing record.

4. The effect of this decision will be to severely restrict the amount of Aldrin-Dieldrin which will be placed into the environment during the 1975 growing season. It will not completely curtail the addition of these compounds into the environment, since the use of existing stocks will be permitted. I am persuaded that permitting the use of this relatively small amount of Aldrin-Dieldrin will be safer environmentally than attempting to retrieve the products, transport them, and then somehow dispose of the consolidated and remaining supplies. In addition, it would not be appropriate to penalize farmers who have already purchased the compounds with the expectation of using them during the remainder of the current growing season. This decision will, however, substantially eliminate the unnecessary or excessive use of Aldrin-Dieldrin in many areas in the 1975 growing season and it will encourage the use of environmentally safer pest control chemicals, as well as other non-chemical pest control methods.

[FIFRA Dockets No. 145 etc.]

SHELL CHEMICAL CO., ET AL.

ORDER OF THE ADMINISTRATOR

In accordance with the foregoing Opinion, the registrations issued under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA), as amended, 7 U.S.C. Sec. 135, et seq., for all pesticide products containing Aldrin or Dieldrin which are subject to and for which appeals were duly filed from the Aldrin-Dieldrin cancellation order issued by the Administrator of the Environmental Protection Agency on June 26, 1972, are hereby suspended and the production for use of all such pesticide products is prohibited. Any stocks of technical grade Aldrin or Dieldrin formulated into products after August 2, 1974, may not be placed in commerce, sold, or used for any purposes other than those specifically exempted in the June 26, 1972 cancellation order, as confirmed in the December 7, 1972 order (see Opinion, p. 6, note 1).

All registrations of Aldrin and Dieldrin held by registrants subject to the Aldrin-Dieldrin cancellation order issued on June 26, 1972 which may be now suspended by operation of law for failure to file timely appeals or objections also are hereby deemed suspended.

Notwithstanding the foregoing, for the reasons stated in my notice of Intention to Suspend dated August 2, 1974, and in accordance with the "Special Rule" provisions of section 15(b)(2) of FIFRA, the continued sale and use of existing stocks of registered products containing Aldrin or Dieldrin which were formulated prior to August 2, 1974 shall be permitted.

Dated: October 1, 1974.

RUSSELL E. TRAIN.

[FR Doc.74-23964 Filed 10-17-74;8:45 am]

[illegible] Shell's post-suspension hearing brief only discusses benefits to corn production (Shell Brief, pp. II–1 thru II–25). It must be presumed that, to the extent Shell defends Aldrin-Dieldrin use on citrus (as well as seed treatment and other minor uses), it relies upon grower testimony given in the cancellation proceeding. USDA has taken the lead on the defense of these uses (See USDA post-hearing Brief, in its entirety, which discusses uses on onions in the Tulelake Basin of Northern California, the strawberry industry in Oregon and Washington, pineapple, sugar cane, and banana production in Puerto Rico, and USDA and state quarantine programs). Shell Brief No. V, p. 6.

[illegible] See Recommended Decision, p. 90; EPA Brief, p. 217.

[illegible] See Recommended Decision, p. 90.

[illegible] See Transcript 2324; 2526–27; 2535–36; 2719; 2720–21.

[illegible] EDF Brief, III–B, p. 31; Florida Citrus Mutual, Ex. 1, p. 1–3.

[illegible] The figure given in the Orlando, Florida, public hearing by Dr. Robert Bullock of the Agricultural Research Center in Fort Pierce, was that 30,000 pounds of Aldrin-Dieldrin were used. He has since informed me by affidavit, dated 17 September 1974, that this testimony was in error and that the correct figure is approximately 120,000 pounds of technical Aldrin. Letter from James T. Griffiths, Florida Citrus Mutual, 20 September 1974, enclosing Dr. Bullock's affidavit.

[illegible] EDF has questioned whether Aldrin-Dieldrin remains effective against citrus pests. The most recent test was conducted 16 years ago by Dr. King, who concluded that Aldrin was only effective 70% of the time. EDF Brief, III–B, p. 32.

[illegible] See, e.g., Transcript (Cancellation) 3468; 3255; 3263.

[illegible] See Transcript (Cancellation) 23766–7; 23791.

[illegible] See Recommended Decision, p. 15.

[illegible] The use of Aldrin-Dieldrin to assure compliance with USDA and state quarantine programs is an especially troublesome point. The existing federal program requires 100 percent control, which is often assumed if Aldrin-Dieldrin is used. There is a substantial question whether the requirement for 100 percent control is necessary or desirable and whether Aldrin-Dieldrin, in fact, even approximates this level. This requirement should be re-examined in light of the findings in this proceeding.

[illegible] See EPA brief, List of Alternative Registrations, pp. 176–79.

Appendix B

FEDERAL REPORTER

Second Series

Volume 510 F.2d

Cases Argued and Determined
in the
UNITED STATES COURTS OF APPEALS
UNITED STATES COURT OF CLAIMS
UNITED STATES COURT OF CUSTOMS
AND PATENT APPEALS
AND
TEMPORARY EMERGENCY COURT OF APPEALS

ST. PAUL, MINN.
WEST PUBLISHING CO.
1975

trial, we think the witness under examination may not be asked to pass upon the mental condition of those artists. To do so raises collateral issues foreign to the trial of appellant. Moreover, the paintings of appellant referred to by his witness, if they can be made available, should be exhibited to the jury for comparison with any paintings of the well-known artists which might be exhibited. Unless these precautions, with any others thought appropriate by the trial judge, are taken, the proceedings become so misleading as to be quite unfair to the jury and, therefore, to appellant. We do not question, however, the right of the prosecution to seek to refute by expert testimony the appropriateness of using the paintings of a patient in aid of diagnosing his mental condition.

When considered together the several difficulties we have noted affecting the second phase of the trial combine to lead us to reverse the verdict on the insanity issue and to remand the case in that respect for further proceedings consistent with this opinion, while affirming the verdict reached on the first phase of the bifurcated trial.

It is so ordered.

SUPPLEMENTAL OPINION

FAHY, Senior Circuit Judge:

The opinion of the court of April 7, 1975, 167 U.S.App.D.C. —, 510 F.2d 1283, 1288, refers to a motion of stipulation proposed to be filed by counsel for appellant correcting, as erroneous, a portion of the transcript of the trial relied upon by the United States as constituting a waiver of appellant's claim of right to a separate jury. Our opinion states, "we have received no stipulation or motion . . . ." The fact is an order of the trial judge correcting the record, based on a stipulation of counsel for the parties, was filed prior to argument of this case, but it was not brought to the attention of the court until subsequent to issuance of the court's opinion April 7, 1975. The stipulation and order confirm the analysis of the transcript in all relevant respects as made by the court in its opinion.

ENVIRONMENTAL DEFENSE FUND, INC., and National Audubon Society, Petitioners,

v.

ENVIRONMENTAL PROTECTION AGENCY and Russell E. Train, Administrator, Respondent,

Shell Chemical Company and Earl L. Butz, Secretary of Agriculture, Intervenor.

SHELL CHEMICAL COMPANY, DIVISION OF SHELL OIL COMPANY, Petitioner,

v.

ENVIRONMENTAL PROTECTION AGENCY and Russell E. Train, Administrator, Environmental Protection Agency, Respondents.

FLORIDA CITRUS MUTUAL, Petitioner,

v.

ENVIRONMENTAL PROTECTION AGENCY and Russell E. Train, Administrator, Environmental Protection Agency, Respondents.

Earl L. BUTZ, Secretary of Agriculture of the United States, Petitioner,

v.

Russell E. TRAIN, Administrator of the Environmental Protection Agency, Respondent.

Nos. 74–1924, 74–2113, 74–2114 and 75–1092.

United States Court of Appeals, District of Columbia Circuit.

Argued Feb. 7, 1975.

Decided April 4, 1975.

On petitions for review of an order of the Environmental Protection Agency suspending the registration and prohibiting the manufacture and sale of the pesticides aldrin and dieldrin, the Court of Appeals, Leventhal, Circuit Judge, held,

Reproduced from best available copy.

inter alia, that (1) the EPA's order was a rational exercise of discretion and supported by the reasoning of the Agency and substantial evidence in the record, and (2) the record failed to establish that the suspension order was tainted by ex parte communications from the Agency's enforcement staff, who were at the time involved in a continuing cancellation hearing; rather, there was no claim of consultation between the prosecutorial and adjudicative staff of the Agency except on the issue of whether to start a suspension proceeding.

Suspension order affirmed, except that the issue of exempting existing stocks of the pesticides remanded for further consideration.

1. Poisons ⇨2

Environmental Protection Agency order suspending the registration and prohibiting the manufacture and sale of the pesticides aldrin and dieldrin was a rationale exercise of discretion and supported by the reasoning of the Agency and substantial evidence in the record. Federal Environmental Pesticide Control Act of 1972, § 6(c), 7 U.S.C.A. § 136d(c).

2. Poisons ⇨2

Environmental Protection Agency's finding that aldrin/dieldrin presents "an imminent hazard during the time required for cancellation" had an adequate evidentiary basis, including scientific data that the pesticides were carcinogenic in mice and rats, and that a causal connection exists between the implantation of the pesticides in the ground soil at the base of plants and the ingestion of pesticide residues by humans. Federal Environmental Pesticide Control Act of 1972, § 6(c), 7 U.S.C.A. § 136d(c).

3. Poisons ⇨2

Within provision of the Federal Environmental Pesticide Control Act permitting the suspension of a registration while a cancellation hearing is pending when "the Administrator determines that action is necessary to prevent an imminent hazard during the time required for cancelation," the term "imminent hazard" is not limited to a concept of crisis; it is enough if there is a substantial likelihood that serious harm will be experienced during the year or two required in any realistic projection of the administrative process. Federal Environmental Pesticide Control Act of 1972, § 6(c), 7 U.S.C.A. § 136d(c).

See publication Words and Phrases for other judicial constructions and definitions.

4. Poisons ⇨2

Federal Environmental Pesticide Control Act does not require the Administrator of the Environmental Protection Agency to establish that a product is unsafe, but places the burden of establishing safety on the applicant and registrant. Federal Environmental Pesticide Control Act of 1972, § 6(c), 7 U.S.C.A. § 136d(c).

5. Poisons ⇨2

Conclusion of the Administrator of the Environmental Protection Agency that the pesticides aldrin and dieldrin present an "imminent hazard" to man because the pesticides are carcinogenic in mice and probably carcinogenic in rats as well was a conclusion within the scientific expertise of the Agency and was not infected by error of law; likewise, the validity of extrapolation to humans from data derived from tests on animals was a matter within the Agency's expertise. Federal Environmental Pesticide Control Act of 1972, § 6(b, c), 7 U.S.C.A. § 136d(b, c).

6. Poisons ⇨2

Although extrapolation of cancer data from mice to man may be quantitatively imprecise, it is sufficient to establish a "substantial likelihood" that harm will result from the use of pesticides found to be carcinogenic in mice. Federal Environmental Pesticide Control Act of 1972, § 6(c), 7 U.S.C.A. § 136d(c).

7. Administrative Law and Procedure ⇨502

An agency is not required to adhere to a prior policy with iron rigidity; all that the law requires is that it explain the reasons for a modification.

8. Poisons 🔑2

His previous refusals on a different record, to suspend the registration of aldrin and dieldrin did not strip the Administrator of the Environmental Protection Agency of discretion to make a policy judgment that, because of new information elicited, the unexpected length of cancellation hearing, and a threat by the manufacturer to commence production of the pesticides for the 1975 season, the risk posed by the registration had increased significantly and suspension had become necessary. Federal Environmental Pesticide Control Act of 1972, § 6(b, c), 7 U.S.C.A. § 136d(b, c).

9. Poisons 🔑2

Record supported Environmental Protection Agency's finding, in support of the suspension of the registration of aldrin and dieldrin, of a "substantial likelihood" that serious harm to man would result from the continued use of the pesticides for the treatment of corn soil insects. Federal Environmental Pesticide Control Act of 1972, § 6(c), 7 U.S.C.A. § 136d(c).

10. Poisons 🔑2

It is not necessary to have evidence on a specific use or area in order to be able to conclude on the basis of substantial evidence that the use of a pesticide in general is hazardous and that the registration of it should therefore be suspended. Federal Environmental Pesticide Control Act of 1972, § 6(c), 7 U.S.C.A. § 136d(c).

11. Poisons 🔑2

Manufacturer of the suspended pesticides aldrin and dieldrin, which were shown to create a risk of cancer in humans, failed to establish that the benefits derived from the pesticides outweighed the harm done by them.

12. Poisons 🔑2

Responsibility to demonstrate that the benefits outweigh the risk is upon the proponents of continued registration of a chemical poison.

13. Poisons 🔑2

Where the Environmental Protection Agency declines to suspend the registration of a chemical poison in the face of evidence of carcinogenicity, it bears the burden of justifying its lack of action; on the other hand, where the Agency decides to act, the burden is on the registrant to establish that continued registration poses no safety threat. Federal Environmental Pesticide Control Act of 1972, § 6(b, c), 7 U.S.C.A. § 136d(b, c).

14. Poisons 🔑2

Conclusion of the Administrator of the Environmental Protection Agency, in support of order suspending the registration of the pesticides aldrin and dieldrin, that alternative methods were sufficiently efficacious in controlling corn pests was supported by the evidence of record; and testimony that carbamate and organophosphate alternatives do not pose the same cancer risk as aldrin and dieldrin supported the finding that available alternatives were environmentally suitable. Federal Environmental Pesticide Control Act of 1972, § 6(b, c), 7 U.S.C.A. § 136d(b, c).

15. Poisons 🔑2

When the subject is risk of cancer, convenience may be relevant but it does not weigh heavily in determining whether the registration of certain pesticides should be suspended. Federal Environmental Pesticide Control Act of 1972, § 6(c), 7 U.S.C.A. § 136d(c).

16. Poisons 🔑2

While a more careful exploration of the availability of alternatives for minor uses would be contemplated for the final determination on the cancellation vel non of the registration of the pesticides aldrin and dieldrin, no such extended discussion of the evidence could be demanded for every use of those pesticides at the emergency, provisional stage involving suspension of the registration. Federal Environmental Pesticide Control Act of 1972, § 6(b, c), 7 U.S.C.A. § 136d(b, c).

17. Poisons 🔑2

Notwithstanding claim that findings of the Administrator of the Environmental Protection Agency were too incomplete to be adequate for an order suspending the registration of aldrin and

Reproduced from best available copy.

dieldrin, it was sufficient, in view of the statutory time constraint under which the Administrator acted, that there was substantial evidence in the record and that the reviewing court was able to discern the fair import of the Administrator's reasoning. Federal Environmental Pesticide Control Act of 1972, § 6(c)(2), 7 U.S.C.A. § 136d(c)(2).

18. Administrative Law and Procedure ⇨676

Where the administrative record is complex and the time for explication brief, judicial review is conducted on the basis of the record as a whole, so that rather conclusory findings can be redeemed by resort to a detailed factual record.

19. Poisons ⇨2

Record failed to establish that Environmental Protection Agency order suspending the registration and prohibiting the manufacture and sale of the pesticides aldrin and dieldrin was tainted by ex parte communications from the Agency's enforcement staff, who were at the time involved in a continuing cancellation hearing; rather, there was no claim of consultation between the Agency's prosecutorial and adjudicative staff, except on the issue of whether to start a suspension proceeding. Federal Environmental Pesticide Control Act of 1972, § 6(b, c), 7 U.S.C.A. § 136d(b, c).

20. Poisons ⇨2

In respect to October, 1974 Environmental Protection Agency order suspending the registration and prohibiting the manufacture and sale of the pesticides aldrin and dieldrin, that part of the order exempting existing stocks of the pesticides would be remanded for further consideration, as the Agency was presented in January of 1975 with estimates that approximately five percent of the total 1974 amount of aldrin granules would be available for use in 1975. Federal Environmental Pesticide Control Act of 1972, § 6(c), 7 U.S.C.A. § 136d(c).

Petitions for review of an order of the Environmental Protection Agency.

William A. Butler, with whom Jacqueline M. Warren, John F. Dienelt, and John T. Shinkle, Washington, D. C., were on the brief for petitioners in No. 74-1924.

Dennis G. Lyons, Washington, D. C., with whom David H. Lloyd, Andrew S. Krulwich, and Linda F. Blumenfeld, Washington, D. C., were on the brief, for petitioner in No. 74-2113 also argued for petitioner in No. 74-2114.

Raymond W. Fullerton, Atty., Dept. of Agriculture, with whom John A. Knebel, Gen. Counsel, James Michael Kelly, Asst. Gen. Counsel, and Richard S. Wasserstrom, Atty., Dept. of Agriculture, were on the brief for petitioner in No. 75-1092.

Charles W. Lane, III, New Orleans, La., was on the brief for petitioner in No. 74-2114.

Michael H. Stein, Atty., Dept. of Justice, with whom Carla A. Hills, Asst. Atty. Gen., Stephen F. Eilperin, Atty., Dept. of Justice, and William E. Reukauf, Atty., Environmental Protection Agency, were on the brief for respondents.

Before WRIGHT and LEVENTHAL, Circuit Judges, and DAVIS,* Judge, United States Court of Claims.

Opinion for the Court filed by Circuit Judge LEVENTHAL.

LEVENTHAL, Circuit Judge:

This case involves the validity of an order issued by the Administrator of the Environmental Protection Agency (EPA) on October 1, 1974, suspending the registration and prohibiting the manufacture and sale[1] of the pesticides aldrin and dieldrin.[2] EPA permitted the sale and use of existing stocks manufactured prior to August 2, 1974, the date of the issuance of the Notice of Intention to Suspend.

---

* Sitting by designation pursuant to 28 U.S.C. § 293(a)

[1] The suspension order covered all pesticide products containing aldrin or dieldrin for which appeals had been filed from EPA's June 26, 1972, notice of cancellation. Certain minor uses—the dipping of roots or tops of non-food plants, subsurface ground insertions for termite control, and mothproofing by those manufacturing processes that utilize the pesticide in a closed system—were exempted

The validity of the suspension of registration is attacked by Shell Chemical Company, the sole United States manufacturer of the pesticides, which raises general questions as to the basis of the order and stresses the importance of these pesticides for the 1975 corn crop. It is also attacked by Florida Citrus Mutual (FCM), an association of citrus growers, and by the Secretary of Agriculture;[3] in addition to adopting the general attack made by Shell, the Secretary stresses the need for the continued registration of aldrin/dieldrin for certain minor uses, including the protection of citrus fruits, onions, strawberries, pineapples, sugar cane, bananas, cranberries, and nursery stock, and use as a seed treatment.

The Environmental Defense Fund (EDF) and the National Audubon Society attack the EPA's decision to permit continued sale and use of existing stocks.

The court has taken into account the need for an expeditious determination, and has, to the extent permitted by its other pressing obligations, expedited the appeal and oral argument, and the issuance of its opinion. While the court has set forth its reasons it has not provided a full elaboration. The court has considered, though it has not spelled out in detail, all the contentions of the various petitioners. It rejects those contentions except that, in the case of the point raised by EDF, the court remands the record for further consideration.

I. THE ORDER

On December 3, 1970, EDF first petitioned EPA for the immediate suspension of aldrin/dieldrin and the initiation of cancellation proceedings for all existing registrations. On March 18, 1971, the Administrator issued notices of intent to cancel, under § 4 of the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA), for all products containing the pesticides, on the basis of his finding that "a substantial question as to the safety" of the chemicals existed.[4] Section 4 permits the suspension of registration while a cancellation hearing is pending when "the Administrator . . . finds that action is necessary to prevent an imminent hazard to the public," but the Administrator declined to take this further step.[5] Registrants objected to the notices of intent to cancel, and requested the appointment of a scientific advisory committee and the commence-

---

by the cancellation order and were not suspended.

Registrants had previously agreed to delete label directions concerning aerial applications and use for control of fire ants, and to withdraw dust formulation registrations.

[2] Aldrin and dieldrin are the common names of two chemical compounds of the chlorinated hydrocarbon family. Aldrin is used in much greater quantities. In 1972, almost twelve million pounds of aldrin were used, while only about 700,000 pounds of dieldrin were used. Joint Appendix in Nos. 74-2113, 74-2114, 75-1092 (J.A.), at 1311. In the soil, aldrin quickly breaks down into dieldrin. The primary use of aldrin and dieldrin today is in the control of corn pests, specifically, the wireworm and the black cutworm. The primary corn pest, the rootworm, is largely resistant.

[3] The Secretary of Agriculture is represented by his own counsel, since the Department of Justice is representing EPA.

[4] The relevant section is now § 6(b) of FIFRA, 7 U.S.C. § 136d(b) (Supp. II, 1972).

" . . . FIFRA requires the Secretary to issue cancellation notices and thereby initiate the administrative process whenever there is a substantial question about the safety of a registered pesticide." Environmental Defense Fund, Inc. v. Ruckelshaus, 142 U.S.App.D.C. 74, 84, 439 F.2d 584, 594 (1971).

[5] Essentially the same standard is now incorporated in § 6(c) of FIFRA, see 7 U.S.C. § 136d(c) (Supp. II, 1972). The Administrator found that "the substantial question of the safety of these registrations is primarily raised by theoretical data, while review of the evidence from the ambient environment indicates that such potential hazards are not imminent in light of the present registrations." EPA, "Reasons Underlying the Registration Decisions Concerning Products Containing DDT, 2, 4, 5-T and Aldrin and Dieldrin," March 18, 1971, J.A. 23.

Reproduced from best available copy.

ment of a public hearing.[6] When EDF sought review in this court of the refusal to suspend, we remanded for further consideration in light of the Report of the Advisory Committee, which was issued on March 28, 1972. Environmental Defense Fund, Inc. v. Environmental Protection Agency, 150 U.S.App.D.C. 348, 465 F.2d 528 (1972). After considering the Report and further public comments, the EPA issued an order on December 7, 1972, which affirmed its previous decisions to issue a notice of intent to cancel, without interim suspension.

Cancellation hearings began before Chief Administrative Law Judge (ALJ) Perlman on August 7, 1973. Twelve months into the hearings, on August 2, 1974, the Administrator issued a notice of intent to suspend on the ground that evidence developed since December 1972 indicated that the continued use of aldrin/dieldrin presented an "imminent hazard" to the public. Shell and USDA requested a public hearing on the suspension question. The hearing began before ALJ Perlman on August 14, 1974, and was concluded on September 12, 1974. ALJ Perlman recommended suspension, and, on October 1, 1974, the Administrator suspended the registrations.

We will first develop the general purpose and validity of the order, with a broad overview of its reasoning and the supporting evidence. Then we shall turn to certain particular objections presented by the parties.

II. GENERAL VALIDITY

[1] Turning first to the broad question of validity raised by cases like this, the court concludes: The EPA's order is a rational exercise of discretion, rather than arbitrary agency action. It is supported by the reasoning of the agency, and by substantial evidence in the record.

A. *The Scope of Judicial Review*

[2] The primary challenge raised by Shell, FCM, and the USDA goes to the adequacy of the evidentiary basis of the EPA's finding that aldrin/dieldrin presents "an imminent hazard [to man] during the time required for cancellation."

[3, 4] We have cautioned that the term "imminent hazard" is not limited to a concept of crisis: "It is enough if there is *substantial likelihood* that serious harm will be experienced during the year or two required in any realistic projection of the administrative process." Environmental Defense Fund, Inc. v. EPA, *supra*, 150 U.S.App.D.C. at 360, 465 F.2d at 540 (emphasis added). "FIFRA confers broad discretion" on the Administrator to find facts and "to set policy in the public interest." Wellford v. Ruckelshaus, 142 U.S.App.D.C. 88, 91, 439 F.2d 598, 601 (1971). *See also* Environmental Defense Fund, Inc. v. EPA, *supra*, 150 U.S.App.D.C. at 354, 465 F.2d at 534 (1972). It does not require the Administrator to establish that the product is unsafe, but places "[t]he burden of establishing the safety of a product requisite for compliance with the labeling requirements . . . at all times on the applicant and registrant." Environmental Defense Fund, Inc. v. EPA, *supra*, 150 U.S.App.D.C. at 352, 465 F.2d at 532.

Section 16(b) of FIFRA defines the scope of judicial review of EPA orders made after public hearing:[7]

> The court shall consider all evidence of record. The order of the Administrator shall be sustained if it is supported

---

[6] Under the terms of the Act applicable at the time, the report of the Advisory Committee was to issue before the commencement of the administrative hearings. § 4(c), 78 Stat. 190 [illegible]64). That provision was amended in 1972 to provide that the hearing examiner could, at his own option or at the request of any party, refer relevant questions of scientific fact to a Committee of the National Academy of Sciences. § 6(d), 7 U.S.C. § 136d(d) (Supp. II, 1972).

[7] If no request for a hearing is made, the suspension order takes effect and is not reviewable by a court. § 6(c)(2), 7 U.S.C. § 136d(c)(2) (Supp. II, 1972).

by substantial evidence when considered on the record as a whole.[8]

The standard of "substantial evidence" means

> something less than the weight of the evidence . . . [T]he possibility of drawing two inconsistent conclusions from the evidence does not prevent an administrative agency's finding from being supported by substantial evidence.[9]

In applying this principle to review of a suspension decision, this court has said, "the function of the suspension decision is to make a preliminary assessment of evidence, and probabilities, not an ultimate resolution of difficult issues. We cannot accept the proposition . . . that the Administrator's findings . . [are] insufficient because controverted by respectable scientific authority. It [is] enough at this stage that the administrative record contain[s] respectable scientific authority supporting the Administrator." Environmental Defense Fund, Inc. v. EPA, *supra*, 150 U.S.App. D.C. at 357, 465 F.2d at 537.

B. *Carcinogenicity of Aldrin/Dieldrin*

Although the cancellation hearing encompasses a broad range of issues concerning the effect of aldrin/dieldrin on the environment as well as on human beings,[10] the supension hearing was confined to whether the pesticides present a cancer hazard to man.[11] The Administrator concluded that aldrin/dieldrin presented an "imminent hazard" to man on the basis of data indicating that it is carcinogenic in five strains of mice and, as corroboration, indications that "there is a *strong probability* that Aldrin-Dieldrin is a carcinogen in rats as well as mice." [12]

1. *Mice Data*

Shell attacks the Administrator's reliance on mice data on the ground that the inadequacy of present knowledge regarding cancer and the difficulty of extrapolating from mice to men render his decision speculative.

[5] The Administrator's failure to determine a threshold level of exposure to aldrin/dieldrin does not render his determination improper, for he has concluded that the concept of a threshold exposure level has no practical significance where carcinogens are concerned. This is due in part to the irreversibility and long latency period of carcinogens. "[W]here the matter involved is as sensitive and fright-laden as cancer," [13] and the statute places the burden on the registrant to establish the safety of his product, we shall not, assuming a substantial showing of danger, require the Administrator to make impossible proofs. In reviewing administrative actions, courts "cannot fairly demand the perfect at the expense of the achievable." [14] The Administrator's conclusion is within the scientific

---

[8] For § 16(b), see 7 U.S.C. § 136n(b) (Supp. II, 1972). In Consolidated Edison Co. v. NLRB, 305 U.S. 197, 229, 59 S.Ct. 206, 217, 83 L.Ed. 126 (1938). Chief Justice Hughes described "substantial evidence" as "more than a mere scintilla. It means such relevant evidence as a reasonable mind might accept as adequate to support a conclusion."

[9] Consolo v. FMC, 383 U.S. 607, 620, 86 S.Ct. 1018, 1026, 16 L.Ed.2d 131 (1966). *See also* Environmental Defense Fund, Inc. v. EPA 160 U.S.App.D.C. 123, 127, 489 F.2d 1247 1251 (1973). "[A] court may [not] displace the [agency's] choice between two fairly conflicting views, even though the court would justifiably have made a different choice had the matter been before it *de novo*." Universal Camera Corp. v. NLRB, 340 U.S. 474, 488 71 S.Ct. 456, 465, 95 L.Ed. 456 (1951).

[10] Testimony on the non-human health effects of aldrin/dieldrin on marine and freshwater aquatic organisms, birds, land mammals, and soil invertebrates was presented at the cancellation proceeding. Administrator's Opinion (A.O.) at 7 n. 1.

[11] A.O. 7.

[12] A.O. 23.

[13] Environmental Defense Fund, Inc. v. EPA, *supra*, 150 U.S.App.D.C. at 358, 465 F.2d at 538.

[14] Public Service Commission v. FPC (Texas Gulf Coast Area Rate Cases), 159 U.S.App. D.C. 172, 196, 487 F.2d 1043, 1067 (1973) (Leventhal, J., dissenting), vacated and remanded, 417 U.S. 964, 94 S.Ct. 3167, 41 L.Ed.2d 1136 (1974).

Reproduced from best available copy.

expertise of the agency, and is not infected by error of law. *Compare* Environmental Defense Fund, Inc. v. Ruckelshaus, *supra*, 142 U.S.App.D.C. at 86, 439 F.2d at 596.

[6] The validity of extrapolation to humans from data derived from tests on animals is also a matter within the agency's expertise. There was testimony before the Administrator to support such extrapolation,[15] and this court has acknowledged the significance of test animal data when cancer is involved.[16] Use of animal data is particularly appropriate where, as here, accurate epidemiological studies cannot be conducted because the virtually universal contamination of humans by residues of aldrin/dieldrin[17] make it impossible to establish an uncontaminated human control group. The long latency period of carcinogens further hinders epidemiological research, d the ethical problems of conducting ncer experiments on human beings are too obvious to require discussion. Although extrapolation of data from mice to men may be quantitatively imprecise, it is sufficient to establish a "substantial likelihood" that harm will result. *Cf.* Society of Plastics Industry, Inc. v. OSHA, 509 F.2d 1301, at 1308 (2d Cir., Jan. 31, 1975).

Shell claims that tests based on mouse data are not substantial evidence, because mouse livers are unusually susceptible to cancer. Still, Shell's data—of statistically significant incidence of malignant liver tumors—were in strains of mice that were, as was noted by the Administrator, unusually resistant to such tumors.[18] In any event, Shell's objections are outweighed by the substantial evidence supporting EPA's determination that mice are not uniquely susceptible to carcinogens, but are, in fact, good predictors of carcinogenic hazard to man. The Administrator found that rodents are particularly useful experimental animals, in part because of the similarity of their response to carcinogens to the response of man, their short lifespan, and our relatively well-developed understanding of the pathological development of tumors in mice and rats. Respected research institutions such as the National Cancer Institute have used mice extensively[19] because they have found mice to be an accurate predictor of cancer in other species.

2. *Conformance with Prior Agency Orders*

[7] Shell stresses EPA's two earlier refusals to suspend the registration of aldrin/dieldrin despite evidence of its carcinogenicity in mice, and attacks the order under review as an unexplained departure from prior agency policy. To begin, an agency is not required to adhere to a prior policy with iron rigidity; all that the law requires is that it explain the reasons for its modification.[20] The doctrine permitting reconsideration has full vitality as to suspension decisions, for here "the administrative process is a continuing one . . . [that] calls for continuing reexamination at significant junctures." Environmental Defense Fund, Inc. v. EPA, *supra*, 150 U.S. App.D.C. at 361, 465 F.2d at 541. The agency's previous determinations are not fixed or permanent policy decisions, but merely earlier stages in an ongoing review and re-evaluation of the evidence.

[8] The EPA's decision makes it clear that what changed here was not EPA's

---

[15] *E. g.*, J.A. 1997 98 (testimony of Dr. Upton); J.A. 916 (testimony of Dr. Heston); J.A. 905 (testimony of Dr. Farber).

[16] Environmental Defense Fund, Inc. v. Ruckelshaus, *supra*, 142 U.S.App.D.C. at 86 n. 41, 39 F.2d at 596 n. 41.

See note 30 *infra* and accompanying text.

[18] A.O. 21.

[19] A.O. 17.

[20] City of Chicago v. FPC, 128 U.S.App.D.C. 107, 115, 385 F.2d 629, 637 (1967), cert. denied, 390 U.S. 945, 88 S.Ct. 1028, 19 L.Ed.2d 1133 (1968); New Castle County Airport Commn. v. CAB, 125 U.S.App.D.C. 268, 270, 371 F.2d 733, 735 (1966), cert. denied, sub nom. Board of Transportation v. CAB, 387 U.S. 930, 87 S.Ct. 2052, 18 L.Ed.2d 991 (1967); Pinellas Broadcasting Co. v. FCC, 97 U.S.App.D.C. 236, 238, 230 F.2d 204, 206, cert. denied, 350 U.S. 1007, 76 S.Ct. 650, 100 L.Ed. 869 (1956).

policy but the nature of the evidence. The EPA decision was supported in part by a re-analysis, by several pathologists, of slides prepared in the course of three studies conducted by the Food and Drug Administration (FDA) in the early 1960's.[21] Re-evaluation of existing data would be adequate in itself to support the modification of a prior decision,[22] but EPA's decision was also based on data that had only recently come to the agency's attention. For instance, improved analytic techniques revealed higher contamination of major food categories by dieldrin than had previously been contemplated.[23] Current results from ongoing projects concerning air sampling and human tissue data were also available. His previous refusals to suspend, on a different record, did not strip the Administrator of the discretion to make a policy judgment that, because of this new information, the unexpected length of the cancellation hearing, and a threat by Shell to commence production of aldrin/dieldrin for the 1975 season, the risk posed by the registration of the pesticides had increased significantly and suspension had become necessary.

3. *Rat Data*

The Administrator cited data that he interpreted as indicating a "strong probability" that aldrin/dieldrin is a carcinogen in rats. The rat data was derived from three tests, two by the FDA and one confirmatory test from Shell's Tunstall laboratories. At least six witnesses reviewing these studies found a carcinogenic effect or a strong probability of one.[24]

Shell characterizes the Administrator's conclusion as a departure from the ALJ that is insufficiently explained. The difference between the ALJ and the Administrator does not concern evidentiary facts, but is rather a difference in policy concerning the program which the facts warrant—peculiarly a matter for the Administrator to determine. The ALJ was "hesitantly unwilling *at this time* to find that dieldrin is conclusively a carcinogen in the rat although there are indications that this is so." [25] The Administrator thought this caution warranted, but, after "an intensive re-examination of the statistics and testimony," concluded "that there is a *strong probability* that Aldrin-Dieldrin is a carcinogen in rats." [26] Thus, both decision-makers appeared to find substantial, if not conclusive, evidence that aldrin/dieldrin is carcinogenic in the rat. The Administrator did not say that the rat data alone resulted in his suspension order. He relied on the rat data as corroborating the finding of substantial likelihood of serious harm based on the various mice experiments. The Administrator considered what the ALJ had said about rats, and his decision to take the rat data into account, as corroborative of the need for the order recommended by the ALJ, was well within his authority.

C. *Causal Connection To Contamination of Man*

Shell further challenges the Administrator's finding that an "imminent hazard" exists on the ground that the Administrator failed to establish a causal connection between the uses of aldrin/dieldrin that Shell defends (primarily implantation in the ground soil at the base of plants) and the ingestion of pesticide residues by humans. Shell claims that human exposure has resulted primarily from spraying, a use that has been discontinued, and points to the lack of aldrin/dieldrin residues in corn grown on treated soil.

---

[21] J.A. 675-78. The EPA also points out that the original pathology had reported the appearance of "morphologically benign" tumors, a finding that would be considered more meaningful today, for the once significant distinction between benign and malignant tumors has lost much of its validity. EPA Br. at 75 n.89.

[22] Bell v. Goddard, 366 F.2d 177, 181 (7th Cir. 1966).

[23] EPA Ex. 38C, p. 3, *cited in* Shell R. Br. at 9 n.12.

[24] EPA Ex. 40, J.A. 448-49 (Dr. Saffiotti); EPA Ex. 42, J.A. 714-21 (Dr. Reuber); EPA Ex. 48, at 3, J.A. 939 (Dr. Firminger); EPA Ex. 52, J.A. 1023 (Dr. Fears); EDF Ex. 33, J.A. 340-41 (Dr. Epstein); J.A. 1991 (testimony of Dr. Farber).

[25] ALJ Recommended Decision, at 56-57.

[26] A.O. 23.

[9] The record supports the EPA's finding of "substantial likelihood" that serious harm will result from the uses defended by Shell.

Treatment of corn soil insects has consistently been the most prominent use of the pesticides since 1955.[27] Corn soil usage accounted for as much as 80 percent of aldrin usage in 1971.[28] Other suspended uses may have accounted for as much as 6 percent of aldrin sales and approximately half of the dieldrin sales.[29] Moreover, the FDA Market Basket Survey has revealed a consistently high, and increasing, incidence of dieldrin residues in meat, fish, poultry, and dairy products.[30] The EPA National Human Monitoring Survey shows dieldrin residues in 96.5 percent, 99.5 percent, and 98.2 per- t of the human fat samples tested ing the years 1970–1972.[31]

EPA's conclusion that the prohibition of the predominant use would reduce the likelihood of increased exposure is not unreasonable. It is supported by the evidence of record as follows: Aldrin/dieldrin are ghly mobile and persistent chemicals that are not lost by dilution in the inorganic components of the environment. The pesticides persist in the soil for several years, where they are absorbed by the roots and transported to the aerial parts of crops, such as soybeans, which are rotated with corn. Many of these products are important feed components for animanls. The pesticide residues are thus incorporated, directly and indirectly, into the milk, meat, poultry, and soy products consumed by humans.

Shell sees inconsistency in EPA's exemption from suspension of the use of soil-implanted aldrin/dieldrin as a termiticide. EPA explains that, when used for this purpose, the pesticide is buried deep beneath the surface of the land, where it remains undisturbed for years. When used for crop protection, however, aldrin/dieldrin is applied to the top few inches of the soil, in lands typically subject to frequent disturbance through plowing and disking.

There is substantial evidence, plainly sufficient to support the suspension order, at least where, as here, the registrant has failed to come forward with proof showing that no causal connection exists. Shell did not even protest the evidence on causal connection in its argument to the ALJ. Nor did it contest the ALJ's finding of a causal relationship in its objections filed with the Administrator. The ALJ's causation findings are the implied assumption of the Administrator's order. His failure to be explicit on the point yields no basis for legal attack, especially in view of the lack of objection on this ground.

D. Minor Uses

[10] Shell, FCM, and USDA claim that the Administrator has failed to show evidence of the existence of an "imminent hazard" and a causal connection for each suspended use of aldrin/dieldrin. They would place the burden on the agency to bring forth material on each crop and each geographical area touched by the suspension order. But "it is not necessary to have evidence on . . . a specific use or area in order to be able to conclude on the basis of substantial evidence that the use of [a pesticide] in general is hazardous." Environmental Defense Fund, Inc. v. EPA, *supra*, 160 U.S.App.D.C. at 130, 489 F.2d at 1254. "Reliance on general data, consideration of laboratory experiments on animals, etc.," has been found to provide a sufficient basis for an order cancelling the registration of a pesticide. *Id.* The

---

[27] Shell Br. at 8. *See also* Shell Ex. 111, J.A. 1310.

.O. 3; Shell Ex. 111, J.A. 1310.

A.O. 3.

EPA Ex. S 7, J.A. 1158 64.

[31] The data show dieldrin residues during these years averaging 0.27 ppm, 0.29 ppm, and 0.24 ppm. EPA Ex. 47 (Dr. Kutz), Tables 1 and 2, J.A. 925 28; EPA Ex. S-15, J.A. 1261.

same principle applies to a suspension proceeding, where the agency makes only a "preliminary assessment of probabilities."

Most of the minor uses either share the critical factor of implantation in the soil close to the ground surface,[32] which identifies causal connection with human ingestion, or take the form of basal and foliar sprays, which may present an even greater risk to man.[33]

E. *Risk-Benefit Analysis*

[11] Shell, FCM and the USDA further challenge the Administrator's finding that the benefits derived from the suspended uses of aldrin/dieldrin do not outweigh the harms done.

[12] The responsibility to demonstrate that the benefits outweigh the risks is upon the proponents of continued registration.[34] The statute places

> a heavy burden on any administrative officer to explain the basis for his decision to permit the continued use of a chemical known to produce cancer in experimental animals.[35]

[13] In our 1972 opinion, Environmental Defense Fund, Inc. v. EPA, *supra*, we said that "a mere recitation of a pesticide's uses does not suffice as an analysis of benefits" where the EPA has refused to initiate suspension proceedings despite evidence of carcinogenicity and a submission that alternative pest control mechanisms exist. We sought a further "elucidation of basis" from the agency to ensure that the evidence of harm was indeed outweighed by benefits flowing from the continued use of the pesticide. Where, as in that case, the agency declines to act in the face of evidence of carcinogenicity it bears the burden of justifying its lack of action:

> By definition, a substantial question of safety exists when notices of cancellation issue. If there is no offsetting claim of any benefit to the public, then the EPA has the burden of showing that the substantial safety question does not pose an "imminent hazard" to the public.

150 U.S.App.D.C. at 359, 465 F.2d at 539. In the present case, in contrast, the agency has decided to act, and the burden is on the registrant to establish that continued registration poses no safety threat.

1. *Use on Corn*

[14] The Administrator's conclusion that alternative methods are sufficiently effıcacious in controlling corn pests is supported by data from studies comparing aldrin/dieldrin treatment of black cutworms and wireworms with other techniques,[36] as well as by the registration of alternatives for these purposes.

[15] The finding that adequate alternatives will be available for the 1975 planting season is supported by evidence that other chemical pesticides are being produced and nonchemical techniques are available. Alternatives are not available in equal volume, say petitioners. However, the Administrator has determined that no pound-for-pound substitution is necessary because aldrin/dieldrin has been overused in the past as a prophylactic measure and because the threat of corn soil insects is greatly reduced at this time—a conclusion supported by the evidence and one that will not be disturbed by this court. Shell protests that the certain post-emergent treatments impose a much greater work burden on the farmer. When the subject is risk of cancer, convenience may be relevant but it does not weigh heavy in the scales.

---

[32] *E.g.*, USDA Ex. 33, at 12 (use of basal sprays on bananas); USDA Ex. 11, at 2 (use of foliar sprays on cranberries), *cited in* EPA Br. at 88 n. 98.

[33] *Compare* Shell Br. at 44-45.

[34] *E.g.*, Environmental Defense Fund, Inc. v. EPA, *supra*, 150 U.S.App.D.C. at 352, 465 F.2d at 532; Environmental Defense Fund, Inc. v. Ruckelshaus, *supra*, 142 U.S.App.D.C. at 82 n. 22, 439 F.2d at 592 n. 22.

[35] Environmental Defense Fund, Inc. v. Ruckelshaus, *supra*, 142 U.S.App.D.C. at 86 n. 41, 439 F.2d at 596 n. 41.

[36] *E. g.*, EPA Ex. 71, J.A. 1121 25; EPA Ex. 61, J.A. 1092-93; EPA Ex. 61, J.A. 1097 98.

Reproduced from best available copy.

We can hardly overturn the Administrator's conclusion that the alternatives were adequate on the ground that he did not give compelling weight to convenience.

Testimony that carbamate and organophospate alternatives do not share the persistence, lipidsolubility, and bioconcentration in animal or human tissues characteristic of aldrin/dieldrin—and thus do not pose the same cancer risk—supports the finding that the alternatives are environmentally suitable.

2. *Minor Uses*

[16] USDA and FCM challenge the Administrator's failure to provide extensive risk-benefit analysis for each crop and each geographical área for which aldrin/dieldrin has been suspended. They fault the order on the grounds that it makes conclusory findings for minor uses without discussing the contrary evidence and that it lacks substantial record evidence to support its rationale. However, the expedited nature of the suspension proceeding imposes limitations on the degree of detail that can be expected from the Administrator's findings at this stage of the administrative process. A more careful exploration of the availability of alternatives for minor uses would be contemplated for the final determination on cancellation *vel non*, but we cannot demand an extended discussion of the evidence for every use at this emergency, provisional stage.

The record evidence as to the relative risks and benefits of each use is a mixed bag, but it provides substantial support for the Administrator's conclusion. The order cites California experience with alternative pesticides that have proved effective against Fuller's Rose Beetle, the Florida citrus pest controlled by aldrin/dieldrin, and there is also evidence that effective foliar sprays will be available for use should an emergency arise during the suspension period. Moreover, aldrin/dieldrin is used on less than 5 percent of the total citrus acreage, and the ALJ noted that much of that use was a kind of "just in case" insurance, applied even in the absence of knowledge that the pest exists in the pertinent grove. As to other crops, the record indicates, for example, that effective registered alternatives are available; that, in the case of pineapples and cranberries, aldrin/dieldrin offer multi-year protection, so the benefits of past applications will continue; and that the present oversupply in the cranberry market diminishes the prospect of hardship from the suspension in regard to that crop.

3. *Heptachlor as an Alternative*

Shell protests that heptachlor/chlordane, pesticides that demonstrate carcinogenicity in mice and are stored in human tissue in the same levels as aldrin/dieldrin, will, in practice, be used in place of the suspended pesticides. Because heptachlor presents an identical cancer risk, Shell argues, the Administrator's suspension of aldrin/dieldrin does not "prevent" an imminent hazard as required by the statute. Heptachlor is also the subject of cancellation proceedings. There is no law that says that all evils must be attacked at the same time and at the same rate. So far as the public interest is concerned, it suffices to note that there is evidence that heptachlor is not available in large amounts comparable to aldrin/dieldrin stocks of past years, so that, in any event, the EPA suspension will achieve a total reduction in the use of harmful pesticides.[37]

III. OTHER CHALLENGES

A. *Challenge to Findings as Incomplete*

Shell and the other petitioners contend that the Administrator's findings are too incomplete to be adequate for a suspension order. This is not a substantial evidence case, they put it, so much as a challenge to the insufficiency of the

---

Approximately 1.5 million additional pounds of heptachlor/chlordane will be available for corn use in 1975, as compared to 7.6 million pounds of aldrin (assuming 1975 aldrin/dieldrin corn use would be the same as the 1974 use). EPA Br. 105.

findings. Time and again petitioners revert to the Administrator's failure to identify explicitly certain contentions, and his disposition thereof, or to his failure to discuss petitioners' evidence.

Under § 6(c)(2) of FIFRA, the Administrator has a maximum of seven days[38] after the ALJ's decision in which to issue his opinion. This time constraint—part of the statutory plan for expedition in reaching suspension decisions—is material in appraising how much Congress contemplated would be required of the Administrator's findings. *Compare* International Harvester Co. v. Ruckelshaus, 155 U.S.App.D.C. 411, 426–27, 478 F.2d 615, 630–31 (1973). The hearing transcript—not including statements by witnesses, exhibits, and over 1,000 pages of briefs—was almost 4,000 pages long,[39] and even longer portions of the cancellation hearing transcript were also incorporated. The USDA's complaint that the Administrator failed to give "attentive consideration" in his discussion of the minor uses to some sixty exhibits and 2,000 pages of testimony by USDA witnesses leads us to comment that we cannot accept the view that the law requires explicit evaluation of all relevant testimony when that is not necessary to deal with the salient grounds of objection and would interpose a barrier that would preclude practical use of the suspension provisions.

[17, 18] Under these circumstances, we think it sufficient that there is substantial evidence in the record and that the court is able to discern the fair import of the Administrator's reasoning.[40] Even in full-dress proceedings without time constraints a court will accept findings that are not wholly articulate, if they can "discern the path" of the agency.[41] What impresses us about the suspension order under review is the Administrator's sensible effort to write an opinion that emphasized his consideration of the issue that most concerned the parties—carcinogenicity. And his discussion and findings on the other contested matters is adequate to satisfy a reviewing court that his decision was not made arbitrarily and capriciously.

Perhaps a paradigm of petitioners' extremism and contentiousness is the objection that the Administrative Procedure Act requires findings of the agency "on all the material issues" and that the Administrator's opinion is not "salvageable" by reference to the discussion of the ALJ.[42]

In our view, it is a matter not for condemnation, as was suggested in argument, but for commendation, that the Administrator's opinion consumes only 45 pages, whereas the ALJ wrote 109 pages. It would have been desirable for the Administrator to have said explicitly what is clearly implicit in and indeed suffuses his entire opinion, that he accepts the ALJ's findings and reasoning except where a difference in commentary is made explicit. But, in the case of a suspension order issued under such time pressure, we cannot stand on ceremony to the extent of vacating the order, or remanding for further findings because this was not recited in so many words.

B. *Procedural Challenges*

[19] We turn, finally, to the point most ardently pressed by Shell at argument, and adopted by the other petitioners, that the EPA Order is tainted by ex parte communications in that members of the agency's enforcement staff, who

---

[38] The Administrator in this case took eight days, but his failure to meet the deadline was not protested by the parties.

[39] A.O. 1.

[40] Where the record is complex and the time for explication brief, we think it particularly important to note that "judicial review is conducted on the basis of the record as a whole, so that rather conclusory findings can . . be redeemed by resort to a detailed factual record." National Air Carrier Ass'n v. CAB, 141 U.S.App.D.C. 31, 41, 436 F.2d 185, 195 (1970).

[41] Greater Boston Television Corp. v. FCC, 143 U.S.App.D.C. 383, 393, 444 F.2d 841, 851 (1970), cert. denied, 403 U.S. 923, 91 S.Ct. 2233, 29 L.Ed.2d 701 (1971).

[42] Shell R. Br. at 2.

were involved in the continuing cancellation hearing at the time, presented arguments to the Administrator favoring the issuance of a Notice of Intent to Suspend. Shell concedes that communications between the prosecutorial and adjudicative staff of an agency are appropriate prior to the initial filing of an administrative complaint, but argues that this rule does not apply where, as here, the communications take place while another phase of the "same case" is underway. We reject Shell's contention.

Suspension and cancellation hearings are separate proceedings in the respect, critical here, that the decisions in the two proceedings are made under different legal standards.[43] To the extent that they are related, we do not find this to be a bar to the kind of communications engaged in here. It may happen that during the course of an agency proceeding against two individuals the "prosecuting" staff discerns from the evidence that proceedings should also be instituted against, or the initial proceeding broadened to include, a third individual. The prosecutorial staff would not be debarred from consulting with the agency head about these steps by the mere fact that a related proceeding was already under way. The same conclusion is applicable where there is no new party but the emerging evidence indicates that a new charge or a broadened charge is appropriate.

Congress has not accepted the view that the possibilities of unfairness require prohibition of an administrative structure that permits the same agency to issue the notice that begins a proceeding and to make the ultimate determination.[44] It has accepted a pragmatic view that the need for effective control by the agency head over the commencement of proceedings requires an ability to conduct consultations in candor with an investigative section on the question whether a notice should be issued and a proceeding begun, and this notwithstanding any residual possibilities of unfairness.

In this case the agency respected the internal separation of functions provided by Congress in its combination of fairness and pragmatism; there is no claim of consultation between the agency's "prosecutors" and the agency head except on the issue whether to issue a new notice—whether to start a suspension proceeding. There is no allegation of communication between "prosecutor" and agency head regarding the final decision in either the cancellation proceeding or the suspension proceeding.[45]

The Administrator's indications in the Notice that he is "persuaded that there exists an 'imminent hazard'", and that he finds that "a situation exists in which the manufacture of Aldrin and Dieldrin during the coming months will be 'likely to result in unreasonable adverse effects' on man and the environment'"[46] do not represent prejudgment of the merits of the decision to suspend. The Administrator was merely making a determination to begin a suspension proceeding under § 6(c)(1) of FIFRA,[47] accompanied by the prefatory findings required by the law, without prejudice in any way to the consideration that would be given to the suspension record and to the result that would be reached in the light of that record. The proponents of continued registration were given a full hearing on their objections.

---

[43] *See* Environmental Defense Fund, Inc. v. EPA, *supra*, 150 U.S.App.D.C. at 357, 465 F.2d at 537; Environmental Defense Fund, Inc. v. Ruckelshaus, *supra*, 142 U.S.App.D.C. at 81, 439 F.2d at 591.

[44] This view was proposed by Messrs. McFarland, Stason and Vanderbilt, in their additional views that accompanied the Report of the Attorney General's Committee on Administrative Procedure. *See* Administrative Procedure in Government Agencies, Sen. Doc. No. 8, 77th Cong., 1st Sess. 203 (1941).

The only agency for which this view was made a legal requirement is the National Labor Relations Board.

[45] The Administrative Procedure Act, 5 U.S.C. § 554(d) (1970), only prohibits participation or advice in the "decision, recommended decision, or agency review."

[46] Notice of Intention to Suspend, J.A. 76.

[47] 7 U.S.C. § 136d(c)(1) (Supp. II, 1972). Section 6(c)(1) provides that a "notice [of intent to suspend] shall include findings pertaining to the question of 'imminent hazard.'"

Compare FTC v. Cinderella Career and Finishing Schools, Inc., 131 U.S.App.D.C. 331, 338, 404 F.2d 1308, 1315 (1968), where this court held that the FTC could issue a press release stating that it found "reason to believe" the law had been violated soon after the issuance of a complaint.

Because the suspension hearing was expedited,[48] the ALJ incorporated nearly 11,000 pages of transcript and more than 350 written exhibits from the cancellation hearing. Shell protests that these procedures made the hearing inadequate to protect its interests in that the EPA prosecutorial staff and EDF were allowed to incorporate the presentation of their medical and benefits case from the more leisurely cancellation proceeding, where they had discovery and subpoena powers, while the proponents of registration, who had not yet reached that phase of their presentation in the cancellation hearing, were compelled to respond in much less time without similar discovery powers. At the time of the suspension notice, Shell had already called 125 witnesses in the cancellation proceeding.[49] Shell did not make a showing that the shortness of the duration of the suspension hearing precluded a fair disposition, or that more time would have been needed by someone seeking, on an expedited basis, to make a presentation in favor of continued registration. Indeed, although Shell was allotted 12 of the 15 days scheduled for the expedited suspension hearing, it used only 8½ of the twelve days, and did not take advantage of ALJ Perlman's offer to permit the presentation without cross-examination of the written statements of witnesses that could not be heard orally within the allotted time.

C. Exemption of Existing Stocks

[20] EDF charges that the EPA's decision to exempt the sale and use of existing stocks of aldrin/dieldrin from the general suspension is arbitrary and capricious. EPA has responded that this decision was based on an assumption that no appreciable and realistically retrievable stocks existed at the time of the order, and that any denial of procedural rights was harmless error. EPA counsel have informed us that EPA was presented in January 1975 with estimates that approximately 5 percent of the total 1974 amount of aldrin granules will be available for use in 1975, and that EPA intends to investigate the matter further, an ongoing re-evaluation that is entirely appropriate.

We affirm the agency's suspension order of October 1, 1974, except for the exemption of the sale and use of existing stocks. The record is remanded for further consideration of that issue.

So ordered.

---

[48] The August 2, 1974, Notice provided for a 15-day hearing, but only 14 of the 15 days were used.

[49] EPA had called 66 witnesses; EDF, 31; USDA, 12; and the user groups, 15.